MATERNAL AND FETAL OUTCOME IN OBESITY COMPLICATING PREGNANCY

A PROSPECTIVE COHORT STUDY

A Dissertation Submitted to

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"MATERNAL AND FETAL OUTCOME IN OBESITY COMPLICATING PREGNANCY-A PROSPECTIVE COHORT STUDY"** is the bonafide work done by **Dr.K.PADMA BHARATHI** at the **Department of Obstetrics and Gynaecology, Government Kilpauk medical College Chennai** from 2016-2018 under the guidance of **Prof Dr.M.S.SORNAM MD,DGO.,** Professor of Obstetrics and Gynaecology, KMCH, Chennai.

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I solemnly declare that this dissertation **"MATERNAL AND FETAL OUTCOME IN OBESITY COMPLICATING PREGNANCY-A PROSPECTIVE COHORT STUDY"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Prof Dr.M.S.SORNAM MD, DGO.,** Professor ,Department of Obstetrics and Gynaecology, Government Kilpauk Medical College and Hospital, Chennai.

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INTRODUCTION

In general, pregnancy in women is considered unique, physiologically normal episode in women's life. However preexisting morbidity of the mother or fetus can complicate pregnancy and as well as those arising during pregnancy and intrapartum make it a high risk one.

"A pregnancy is defined as high risk, when the probability of an adverse outcome for the mother or child is increased over the base line risk of that outcome among the general population by the presence of one or more ascertainable risk factors."

"One such pre-existing maternal morbidity that makes a pregnancy high risk is obesity". The magnitude of the obesity prevalence has been increasing in developed and developing nations, though in varying degrees. Also coming with the increase in obesity prevalence, inevitably, are the morbidities obesity promotes, including cardiovascular disease, diabetes, hypertension, stroke etc. It becomes a major issue when it affects the women of reproductive age group, as obesity makes a pregnancy high risk, by the increased incidence of gestational diabetes, pre-eclampsia, gestational hypertension, labour induction, increased cesarean rates, anaesthetic complications, postoperative morbidity,

prolonged hospital stay etc.. They are at increased risk of delivering large babies and NICU admission.

Although routine weighing of pregnant women is being carried out in most of the antenatal clinics, not much of importance is given to the weight of the women as such. In fact prenatal counseling plays a vital role in identifying women who are obese. Advice on weight reduction before embarking on pregnancy will go a long way in reducing the morbidity due to obesity in pregnancy.

REVIEW OF LITERATURE

WORLD WIDE PREVALENCE:

For a number of years, obesity has been termed epidemic, strictly defined, the word epidemic implies a temporary wide spread outbreak of greatly increased frequency. Therefore obesity more currently is endemic, a condition that is habitually present. Its prevalence is increasing world wide in both developed and developing countries.

In USA, from 1960-1991 NHANES documented an alarming increase among the adults over the past decades. More than 127 million American adults were over weight,60 million were obese and 9 million were severely obese. Among the women in 1999 through 2000,62% were overweight,34% were obese,6% were severely obese.

The incidence of obesity in pregnancy has increased in concordance with the prevalence in the general US population. The reported incidence of obesity during pregnancy varies between 6% and 28% depending on the obesity definition, year and characteristics of the study population.

Further more, Lu et al examined the longitudinal trend of maternal obesity spanning from 1980-1999.They demonstrated that the incidence

of obesity at the first prenatal visit increased from 7.3% to 24.4% in this 20 years time period.

Increasing prevalence of obesity during 20 years in pregnant women classified at the time of their first prenatal visit at the University of Alabama at Birmingham. (From Lu and colleagues,2001 with permission)

In a study by Glady et al, the largest proportion of obese was among American Africans 22% followed by Latins 14%,Whites 8% and the Asians 4%.

In India a study conducted by Mohan et al at Chennai in 2001 the prevalence in age group more than 20 years was 22.5% males and 31.8% in females.

The various studies conducted in India are shown below:

Prevalence of Obesity in India

Author	City centre	Year	Age (yr)	Prevalence of Obesity(%)	
				Male	female
Dhurandhar & Kulkarni	Bombay	1992	31-50	10.7-53.1	-
Gopinath et al	Delhi	1994	25-64	21.3	33.4
Zargar et al	Kashmir	2000	>40	7.0	23.7
Gopalan	Nutrition foundation of India	1998	-	32.2 16.2 7.0	50 30.3 27.8
National family health survey	-	1998-99	15-49	-	2.3
Mohan et al	Chennai urban population study	2001	>20	22.8 21.5	31.8 36.5
Deshmukh et al	Rural wardha	2006	>18	5.1 7.6	5.2 8.7

Definition of obesity:

"Obesity may be defined as an abnormal growth of the adipose tissue due to enlargement of fat cell or increase in fat cell number or

both". A number of systems have been used to define and classify obesity.

Assessment of obesity:

Although obesity can be easily identified at first sight, a precise assessment requires measurement and reference standards. Various methods to assess the obesity are as follows,

Body weight:

Body weight though not an accurate measure of examining fat, is a widely used index. The various indices used are:

1. **Body mass index-BMI(Quetelet's index)**

   Weight (kg)/height(m2)

2. **Ponderal index**

   Height (cms)/cube root of body weight(kg)

3. **Broca's index**

   Height (cm)-100

4. **Lorentz's formula:**

   Height (cm)-100-Height(cm)-150/2(women)or 4(men)

5. **Other methods:**

   Skin fold thickness

   Waist circumference and waist:hip ratio

USE OF BODY MASS INDEX TO CLASSIFY OBESITY:

BMI is a simple index of weight for height that is commonly used to identify underweight, overweight and obesity in adults.

"It is defined as weight in kgs divided by the square of the height in metres(kg/m2)".

The classification is as follows according to WHO and National Heart Lung and Blood institute(1998).

CATOGORY	**BMI**
Underweight	<18.5(kg/m2)
Normal weight	18.5-24.99(kg/m2)
Over weight	25-29.99(kg/m2)
Obese	>30(kg/m2)

It is believed to be a superior measure of adiposity than weight for height, but it too has limitations, that it does not incorporate a direct measure of body fat composition and distribution.

According to Freedman and colleagues 2002 obesity is further classified as:

CATEGORY	**BMI**
Class I (moderate obesity)	30-34.9(kg/m2)
Class II (severe obesity)	35-39.9(kg/m2)
Class III (very severe obesity)	>40(kg/m2)

EPIDEMIOLOGICAL FACTORS

The etiology of obesity is complex and is one of multiple causation.

Age :

Obesity can occur at any age but generally increase with age.

Childhood obesity:

Infants with excessive weight gain have an increased incidence of obesity in later life. One third of obese adults have been so since childhood.

Sex:

Women generally have higher rate of obesity than men, although men have higher rate of overweight.

Pregnancy and parity:

It has been claimed that women's BMI increases with successive pregnancy. The evidence suggested that this increase is likely to be about 1 kg/pregnancy. Hence multiparous women are obese when compared to nulliparous women.

Genetic factors:

There is a genetic component in the etiology of obesity.

Physical inactivity:

Sedentary life style particularly sedentary occupation and inactive recreation promote it. Physical inactivity may cause obesity which in turn restricts activity. This is a vicious cycle.

Socioeconomic status:

Inverse relationship between socioeconomic status and obesity exist.

Eating habits:

Eating in between meals, preference in sweets, refined foods and fats composition of the diet, periodicity with which it is eaten and the energy derived from it are all relevant to the etiology of obesity.

Psychosocial factors:

Psychosocial factors are deeply involved in the etiology of obesity. Overeating may be a symptom of depression, anxiety, frustration.

Familial tendency:

Obesity frequently run in families.

Endocrine factors:

These factors may be involved in occasional cases.

Eg: Cushing's syndrome, growth hormone deficiency, hypothyroidism

Alcohol:

The relationship between alcohol and adiposity is positive for men and negative for women.

Education:

In affluent countries, inverse relationship between education and prevalence of obesity is seen.

Smoking:

Use of tobacco is reported to lower body weight.

Ethnicity:

Ethnic groups in many industrialized countries appear to be especially susceptible to the development of obesity and its complications. This may be due to genetic predisposition.

Drugs:

Use of certain drugs e.g: Corticosteroids, Contraceptives, Insulin, Beta blockers can promote weight gain.

HAZARDS OF OBESITY

Metabolic syndrome:

Obesity interacts with inherited factors and leads to the onset of insulin resistance. This metabolic abnormality in turn is responsible for altered glucose metabolism and a predisposition to type 2 diabetes and

cardio vascular diseases and accelerate its course. The most important are type 2 diabetes, dyslipidemia and hypertension. Prevalence is increased with age. According to NHANES III, prevalence was about 6% in those with 20 yrs of age,14% in those with 30-39 yrs of age,20% in those with 40-49 yrs of age and >30% for women over 50 yrs of age.

"20% in reproductive age group"

Other complications:

- Obesity cardiomyopathy
- Sleep apnea
- Ischemic stroke
- Gall bladder disease
- Sub fertility
- Carcinoma endometrium
- Deep vein thrombosis
- Poor wound healing

OBESITY IN PREGNANCY:

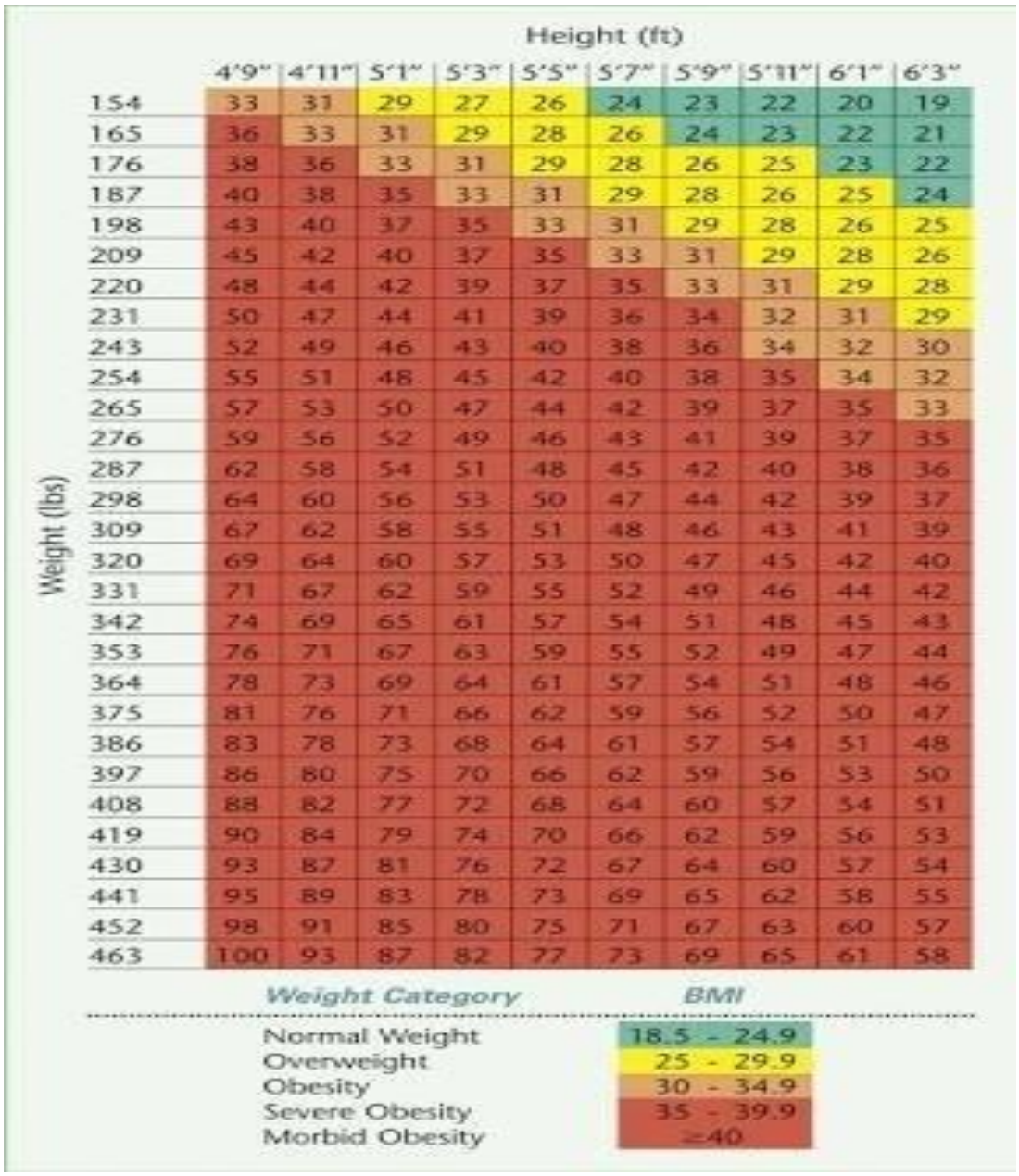
Definition:

In the past obesity in pregnancy was defined using various approaches.

In an effort to provide guidance on this issue in 1990 the Institute of medicine (IOM) recommended that, the BMI be used to define maternal weight groups. In 1993 the ACOG released its BMI classification of maternal weight and optimal weight gain during pregnancy. As a result BMI now serves as a standardized means of evaluating the prevalence and outcomes of obesity during pregnancy.

Diagnosis:

For practical purposes, it is useful to keep in AN clinic, acceptable statistical tables which indicates the BMI for various heights and weights. One such calculated BMI values available in graphic form is shown here.



Height (ft)

Weight (lbs)	4'9"	4'11"	5'1"	5'3"	5'5"	5'7"	5'9"	5'11"	6'1"	6'3"
154	33	31	29	27	26	24	23	22	20	19
165	36	33	31	29	28	26	24	23	22	21
176	38	36	33	31	29	28	26	25	23	22
187	40	38	35	33	31	29	28	26	25	24
198	43	40	37	35	33	31	29	28	26	25
209	45	42	40	37	35	33	31	29	28	26
220	48	44	42	39	37	35	33	31	29	28
231	50	47	44	41	39	36	34	32	31	29
243	52	49	46	43	40	38	36	34	32	30
254	55	51	48	45	42	40	38	35	34	32
265	57	53	50	47	44	42	39	37	35	33
276	59	56	52	49	46	43	41	39	37	35
287	62	58	54	51	48	45	42	40	38	36
298	64	60	56	53	50	47	44	42	39	37
309	67	62	58	55	51	48	46	43	41	39
320	69	64	60	57	53	50	47	45	42	40
331	71	67	62	59	55	52	49	46	44	42
342	74	69	65	61	57	54	51	48	45	43
353	76	71	67	63	59	55	52	49	47	44
364	78	73	69	64	61	57	54	51	48	46
375	81	76	71	66	62	59	56	52	50	47
386	83	78	73	68	64	61	57	54	51	48
397	86	80	75	70	66	62	59	56	53	50
408	88	82	77	72	68	64	60	57	54	51
419	90	84	79	74	70	66	62	59	56	53
430	93	87	81	76	72	67	64	60	57	54
441	95	89	83	78	73	69	65	62	58	55
452	98	91	85	80	75	71	67	63	60	57
463	100	93	87	82	77	73	69	65	61	58

Weight Category	BMI
Normal Weight	18.5 - 24.9
Overweight	25 - 29.9
Obesity	30 - 34.9
Severe Obesity	35 - 39.9
Morbid Obesity	≥40

PROFORMA

NAME :

AGE :

IP NO :

PHONE NUMBER :

ADDRESS :

HUSBANDS NAME :

OCCUPATION :

QUALIFICATION :

SOCIOECONOMIC STATUS :

BOOKING :

IMMUNISATION :

H/O PRESENT ILLNESS :

MENSTRUAL HISTORY : regular/irregular

LMP :

EDD :

MARITAL HISTORY : Married since

Consangunity :

h/o infertility :

OBSTETRIC HISTORY :G P L A

Last child birth

EFFECT OF OBESITY ON PREGNANCY

ANTEPARTUM COMPLICATIONS

Sub fertility:

When considering the impact of obesity on pregnancy it is first important to note that obesity can be a barrier to reproduction. Several studies have reported an association between BMI and infertility, which in the obese infertile women is mainly due to increased insulin resistance and related to amenorrhea and ovulatory dysfunction. In their review, Neill and Nelson-Piercy 2001 linked impaired fecundity in women with BMI>30 kg/m2.

In addition obesity has been associated with an increased risk of spontaneous abortion in patients who receive infertility treatment. However obesity does not appear to be a risk factor for abortion in spontaneously conceived pregnancy.

Pre-pregnancy medical disorders:

Due to their strong association with obesity in the general population Essential hypertension and Diabetes mellitus are the two most common medical complications of obese gravida. Other obesity

associated morbidities such as Coronary heart disease, stroke and cancer have a low prevalence in the reproductive age group.

Obstructive sleep apnea is a rare but serious obesity related morbidity. Data on this complication during pregnancy though limited suggested that obstructive sleep apnea may be precipitated or exacerbated during pregnancy and may be associated with hypertensive disorders during pregnancy and impaired fetal growth.

Pregnancy specific complications:

Gestational diabetes:

Maternal obesity is associated with an increased risk of gestational diabetes. Incidence varies from 7% to 17%.This increased risk is primarily related to an exaggerated increase in insulin resistance in the obese state. An estimate of the incidence of gestational diabetes in the pregnancies of obese gravidas can be derived from the data of Gross et al and Ehrenberg et al who each reported a 6.5% and 8% incidence of gestational diabetes, respectively in obese gravidas who were from a geographically similar U.S urban population. In addition ,the magnitude of this risk is positively correlated with increase in maternal weight. The glucose intolerance associated with gestational diabetes generally

resolves after pregnancy. However women who are obese during pregnancy and develop gestational diabetes have been shown to have a 2-fold increased prevalence of subsequent type 2 diabetes as compared to lean women. Therefore maternal obesity is a significant long term risk factor for type 2 diabetes.

Hypertensive disorders:

The association between obesity and hypertensive disorders during pregnancy has been a consistent finding in the obstetrical literature. Specifically, maternal weight and BMI have been validated as independent risk factors for pre-eclampsia. Sibai et al reported a significant difference in the incidence of pre-eclampsia for women with an early second trimester BMI<20 kg/m2(4.3%)as compared to when the BMI was >34 kg/m2(12.6%,P<0.0001).The mechanism by which obesity imparts an increased resistance and subclinical inflammation and endothelial dysfunction are also responsible for the increased incidence of pre-eclampsia in obese gravidas.

Preterm birth:

Conflicting data exist regarding the relationship between maternal obesity and the risk for preterm birth. Naeye in an analysis of data from

the Colloborative Perinatal study undertaken from 1959 through 1966,reported an increasing incidence of preterm birth between 24 to 34 weeks gestation associated with increasing maternal pregravid body weight. The increased incidence of preterm birth was attributed to an increased prevalence of chorioamnionitis and twin gestations in the higher maternal weight groups. In a more recent population based cohort analysis of Washington state birth certificates, Baeten et al reported an increased risk for preterm birth <32 weeks for women with a pre-pregnancy BMI >30 kg/m2,which remained significant when women without antenatal complications were analysed separately (odd's ratio=1.5)

In contrast, in a larger population based cohort study from Sweden, Cnattingius et al reported an overall increased risk for preterm birth <32 weeks in nulliparas with a BMI>30 kg/m2,but this risk was no longer significant when women with hypertensive disease were excluded. Similarly in a large population based cohort study from England Sebire et al reported no association between BMI and preterm birth <32 weeks when analysis were adjusted for antepartum complications. These data suggest that the increased risk of preterm birth in obese gravidas is primarily associated with obesity related medical and antenatal

complications and not some intrinsic predisposition to spontaneous preterm birth.

Prolonged pregnancy:

There is a growing body of evidence to support the association between obesity and prolonged pregnancy. Although early reports by Calandra et al and Gross et al failed to identify an association between maternal obesity and the incidence of post term (>42 weeks) pregnancy, Johnson et al subsequently reported an independent association between increasing maternal pre-pregnancy weight and BMI and the risk for post term pregnancy. More recently in 2 large cohort studies, Ehrenberg et al reported an increased risk for prolonged pregnancy among obese gravidas (Odd's ratio=1.5) as did Sebire et al (Odd's ratio=1.72)

Multifetal gestation:

An increased incidence of multifetal gestation has been reported among obese gravidas. (Gross et al,Naeye)

Urinary tract infection:

In a pooled analysis of 3 studies, Abrams et al reported that being overweight prior to pregnancy was associated with a 42% increased risk

for urinary tract infections. Its findings have been substantiated by Sebire et al.

Others:

There is no evidence to support an increased risk of abruption placenta or placenta previa (Wolfe HM et al,1994).But results of other studies (Bainco et al,1998) are conflicting.

Ultrasound in obese pregnant mothers:

Obesity can limit the prenatal diagnosis of congenital malformations. Wolfe et al studied the relationship between BMI and the visualization of fetal anatomy. Although obesity poses a significant challenge to the obstetrical sonographer in the diagnosis of fetal malformations, it does not seem to hinder sonographic estimations of fetal weight.

Practical difficulties:

1. Clinical diagnosis of pregnancy is sometimes difficult

2. As pregnancy proceeds it is difficult to evaluate size of the uterus, weight of the uterus, to determine the presenting part, to detect fetal heart sound, presence or absence of hydromnios.

3. Maternal blood pressure is difficult to determine using standard cuff and may show artificially high blood pressure.

4. Difficulty in sonographic visualization in women (Wolfe et al 1990)

5. Cephalo pelvic relationships are difficult to estimate in obese women but potential risk is always present, particularly as multiparity and increased lordosis caused by obesity are both predeterminants of spondylolisthesis.

6. Dyspnea due to exertion

7. Placing of intravenous lines may be difficult

8. Difficulty in monitoring maternal and fetal well being can occur.

Intrapartum complications

Labour Induction:

Understanding of the relationships between obesity and labour characteristics is evolving. Obese gravidas were known to have an increased incidence of labour induction. Estimates of the magnitude of this risk range from a 1.7 fold to 3.3 fold increase which remains

significant even after adjustment for associated antepartum complications.

Dysfunctional labour:

Investigations on the labour characteristics of the obese gravidas are limited and conflicting. Gross et al found no difference in the major dysfunctional labour patterns between obese and non obese parturients. Ekblad et al also found no difference in the duration of the first and second stage of labour between obese parturients or those with excessive weight gain and controls. However Johnson et al reported a higher risk for labour abnormalities with both increasing prepregnancy BMI and gestational weight gain.

Cesarean delivery:

The primary intrapartum complication of obesity is an increased risk for cesarean delivery. Both pre-pregnancy obesity and excessive maternal weight gain contribute to an increased cesarean risk. Importantly these associations appear to be independent of obesity related antenatal complications, short maternal stature, higher infant birth weights and gestational age at delivery. The factors that contribute to obesity related increased cesarean risks are not clear. In a large

population based cohort study of nulliparas conducted in Sweden, Cnattingius et al demonstrated the cesarean rates increased consistently with decreasing maternal height and increasing prepregnancy BMI. Subsequently Young et al reported that among a large cohort of nulliparous women the obesity related increase in cesarean was primarily mediated through an increase in cesarean for cephalopelvic disproportion, failure to progress, which was independent of maternal height. As previously discussed, there is a lack of consistent evidence to support a higher incidence of specific dysfunctional labour patterns among obese parturients. These preliminary data therefore suggest that obesity may lead to dystocia due to increased soft tissue deposition of the pelvis.

Intraoperative complications:

Cesarean in the obese gravida is more often performed emergently and is associated with prolonged incision to delivery interval, blood loss >1000ml,longer operative times and difficulty in delivering the baby.

Skin incision:

Pfannenstiel incision are believed to provide a more secure wound closure and less postoperative pain which can lead to early ambulation and improved respiratory function.

Anesthetic complications:

Increased subcutaneous fat increases the difficulty in placing regional anesthesia and increases the rate of placement failure and thus the need for general anesthesia. Ranta et al reported that obese parturient experience more technical problems in establishing epidural anesthesia, such as inadvertent dural puncture, multiple attempts at placement and senior anesthetist consultation, but experienced an equal response to pain treatment. The greater incidence of medical and antenatal complications, increased risk of cesarean section and higher incidence of anesthetic complications necessitates timely anesthetic evaluation in all obese parturient.

Others:

Investigations that controlled for birth weight ,the incidence of intrapartum complications such as shoulder dystocia, malpresentation, hemorrhage and 4^{th} degree laceration did not appear to increase in obese gravida. However because maternal obesity is a risk factor for fetal macrosomia, the clinician should still anticipate these complications. An increased incidence of intrapartum fetal heart rate abnormalities, cord accidents and meconium stained amniotic fluid has been associated with maternal obesity.

Wound complications:

Obese women have increased rates of wound infection and wound dehiscence. Myles et al found that obesity was an independent risk factor for post cesarean morbidity in women.

Postpartum complications:

Whether delivered vaginally or by cesarean the obese gravida is at higher risk of postpartum endomyometritis, laceration/episiotomy infection and wound infection. Several studies reported a lack of association between postpartum hemorrhage and maternal obesity.

Lactation dysfunction may be another postpartum complication of obesity. Study results are conflicting.

The cumulative effect of obesity related complications during the postpartum period is a resultant prolongation of hospitalization .

Prolonged hospitalization for the obese gravida ultimately translates into increased health care costs.

Perinatal outcome

Birth weight:

Pre-pregnancy obesity and maternal weight gain both plays an important role in determining infant birth weight. Also gestational diabetes is complicated by excessive numbers of large for gestational age and macrosomic infants. As a result the obese gravida is at increased risk of delivering a high birth weight infant.

Anamolies:

There has been an accumulation of evidence to support that maternal obesity is associated with an increased risk of congenital malformations .Watkins et al found that the offspring of obese women have 2 fold increased risk for neural tube defects.

Other malformations are heart defects, ventral wall defects and orofacial defects.

Morbidity and mortality:

Two important and inter-related co factors that contribute to excessive perinatal morbidity and mortality are chronic hypertension and diabetes mellitus both of which are associated with obesity. Chronic

hypertension is a well known cause of fetal growth restriction. Pre-gestational diabetes increases the rate of birth defects. The obesity related preterm birth accounted for nearly half of the mortality.

More recent studies also suggested that obesity is associated with an increased risk of still birth 1.4 to 2.6 fold increased risk for fetal death.

A final outcome to consider is the potential impact of maternal obesity and weight gain on subsequent childhood obesity.

Contraception:

Oral contraceptive pill failure is more likely in overweight women. According to Holt and colleagues 2002 women in the highest weight quartile had 16 fold increased risk of pregnancy. Women who used very low dose OCP had 4-5 fold increase in pregnancy rate.

Long term consequences:

It is intuitive that excessive pre-pregnancy weight can be used to predict long term obesity with its attendant morbidity and mortality. Rooney and Schauberger 2002 however reported the excess weight gain during pregnancy but not pre-pregnancy-is a predictor of long term obesity.

AIMS AND OBJECTIVES

To evaluate the effect of obesity on the maternal and perinatal outcome in pregnancies complicated by obesity

MATERIALS & METHODS

TYPE OF STUDY

- Prospective cohort study

PERIOD OF STUDY

- 7 months (Mar 2017 to Sep 2017)

PLACE OF STUDY

- Antenatal outpatient department& Antenatal Ward
- Labour ward

  Dept. of Obstetrics &Gynaecology

  Govt. Kilpauk Medical College &Hospital,

  Chennai 10.

INCLUSION CRITERIA

- Pregnant women with first trimester BMI>30 kg/m2
- Pregnant women with first trimester BMI between 18.5 kg/m2 and 24.9 kg/m2
- Irrespective of age, parity, socio-economic status.

EXCLUSION CRITERIA

- Mothers not booked at first trimester.
- Miscarriage
- Anomalous baby
- Women with BMI between 25 kg/m2 and 29.9 kg/m2
- Women with BMI <18.5 kg/m2
- Women who could not be followed until delivery.

SAMPLE SIZE & SAMPLING

34 Obese, 34 normal women in first trimester. KMC has on an average 1st trimester AN Registrations of 60 / week. From this I would collect consecutive cases of obese and normal pregnancies till required sample size is reached. At the most it requires 2-3 weeks for recruiting patients. The latest recruited person will be followed for 28 weeks.

SAMPLE SIZE:

- Two sided significance(1-alpha) : 95
- Power(1-beta,%chance of detecting) : 80
- Ratio of sample size,unexposed/exposed : 1
- Percent of unexposed with outcome

  GDM : 5
- Percent of exposed with outcome : 27
- Odds ratio : 8.6
- Risk/prevalence ratio : 5.4
- Risk/prevalence difference : 22

Kelsey

- Sample size-Exposed : 34
- Sample size-Unexposed : 34
- Total sample size : 68

Ref: Diabetes Care 2007 Aug;30(8):2007-6 Epub 2007 April 6

MATERIAL AND METHODS

The present study is carried out as a prospective cohort study and the antenatal mothers are selected according to the criteria and in all women detailed history followed by complete general and physical examination done. Relevant hematological, biochemical investigations, USG also done.

They are followed up to delivery and postpartum until discharge and the following outcomes are studied,

- Gestational diabetes
- Pre-eclampsia
- Gestational hypertension
- Malpresentation
- Multiple pregnancy
- Abruptio placenta
- Placenta previa
- Labour induction and their indication

- Mode of delivery(vaginal/Cesarean delivery)
- Shoulder dystocia
- Instrumental delivery
- Postpartum hemorrhage
- Deep vein thrombosis
- Post op wound infection/dehiscence
- Duration of hospital stay

For Neonates the following outcomes are studied,

- Gestational age at birth,
- Birth weight,
- APGAR at 5 mts,
- Admission in NICU and indications for admission are analysed.

BENEFITS OF THE STUDY

1) Pregnancies among obese women are classified as high risk pregnancies and appropriate antenatal care can be provided with heightened surveillance.

2) Through this study we can anticipate the complications and intervene earlier if complications arise.

3) Obesity is modifiable and preventable so we can give reconception counseling and awareness regarding exercise and healthy nutritious diet.

4) Screening for diabetes and hypertension can be done before conception and at 1st antenatal visit.

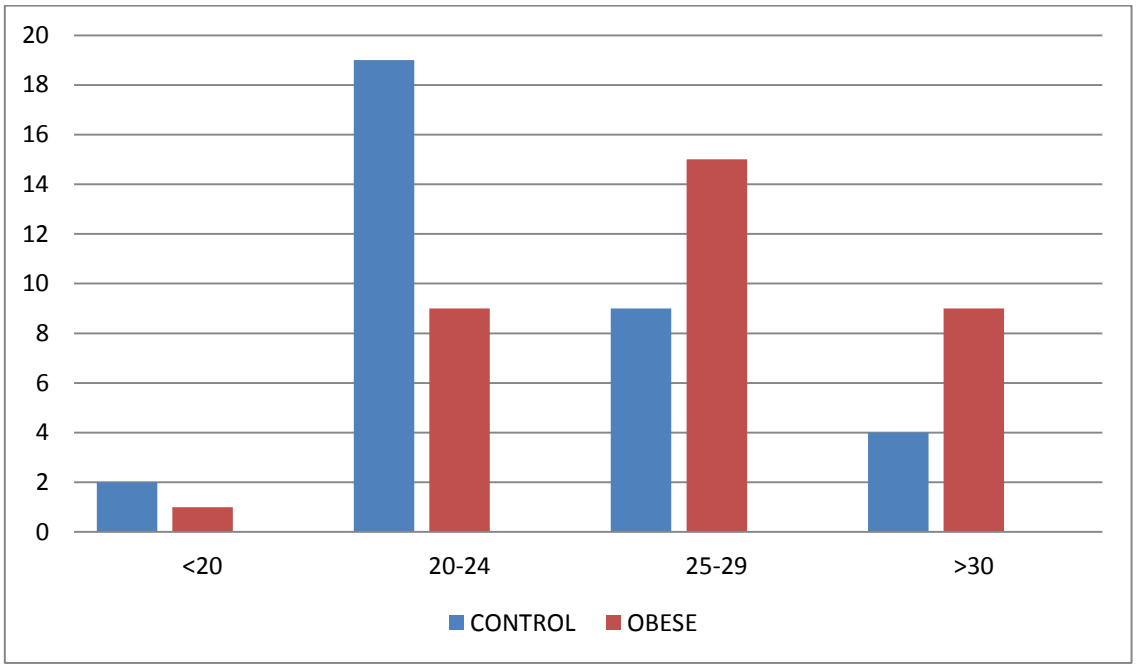
STATISTICAL ANALYSIS

Differences between groups were evaluated using Chi-square and student t test and statistical significance was deemed at a p value of <0.05.Odd's ratio was calculated expressing the relationship between obesity group and specific maternal outcomes.

DATA ANALYSIS

Thirty four pregnant women with BMI >30 kg/m2 and thirty four women with BMI 18.5 kg/m2 to 24.9 kg/m2 were selected and were followed prospectively. The obese with BMI > 30 kg/m2 and control women with normal BMI were studied.

MATERNAL AGE DISTRIBUTION



MATERNAL AGE DISTRIBUTION

AGE(YEARS)	CONTROL		OBESE	
	No	Percentage	No	Percentage
<20	2	5.88%	1	2.94%
20-24	19	55.88%	9	26.47%
25-29	9	26.47%	15	44.11%
>30	4	11.76%	9	26.47%

P<0.05 (significant)

The majority of obese women(44.11%) were between 25-29 yrs where as majority of control women(55.88%) were between 20-24 yrs.proportion of women in the age group>30 yrs were 26.47% in obese groupand only 11.76% in control group.This difference in age group distribution was statistically significant.

AGE IN YEARS

Group	Total	Mean years	Standard deviation	Student t-test
Control	34	24.14	3.424	T=6.12
Obese	34	27.01	4.525	P=0.001

The mean age in obese group was 27.01 yrs where as in control group it was 24.14 yrs(P=0.001).Obese women tend to be older.

MATERNAL WEIGHT

	Group	Total	Mean (kg)	Standard deviation	Student t-test
Wt at booking	Control Obese	34 34	51.25 76.73	4.682 9.065	T=32.1 P=0.001
BMI at booking	Control Obese	34 34	21.7 32.73	1.708 2.662	T=43.3 P=0.001
Wt at delivery	Control Obese	34 34	61.33 83.94	5.602 9.056	T=26.6 P=0.001

The mean weight at booking in obese women was 76.73kg and in control women it was 51.25 kg. The mean BMI at booking in obese women was 32.73 kg/m2 and in control women it was 21.7 kg/m2. The mean weight at term in obese women was 83.94 kg and in control women it was 61.33 kg.

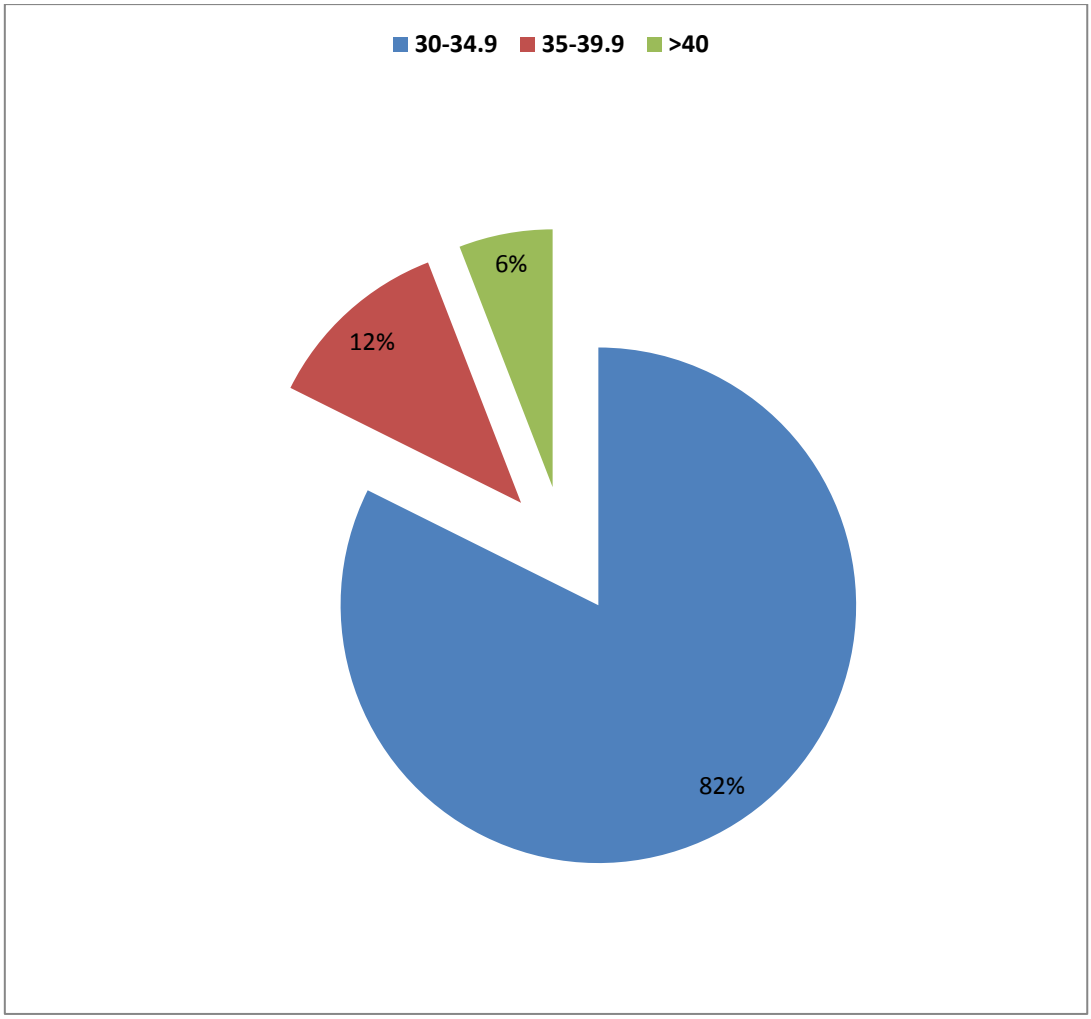
SOCIOECONOMIC STATUS

Socioeconomic class	Control		Obese	
	No	Percentage	No	Percentage
I	-	-	-	-
II	1	2.94%	1	2.94%
III	2	5.88%	4	11.76%
IV	11	32.35%	12	35.29%
V	20	58.82%	17	50%

X2=5.61,P>0.05 not significant

Most of the women in obese and control groups belonged to class V.

CATAGORISATION OF OBESE WOMEN

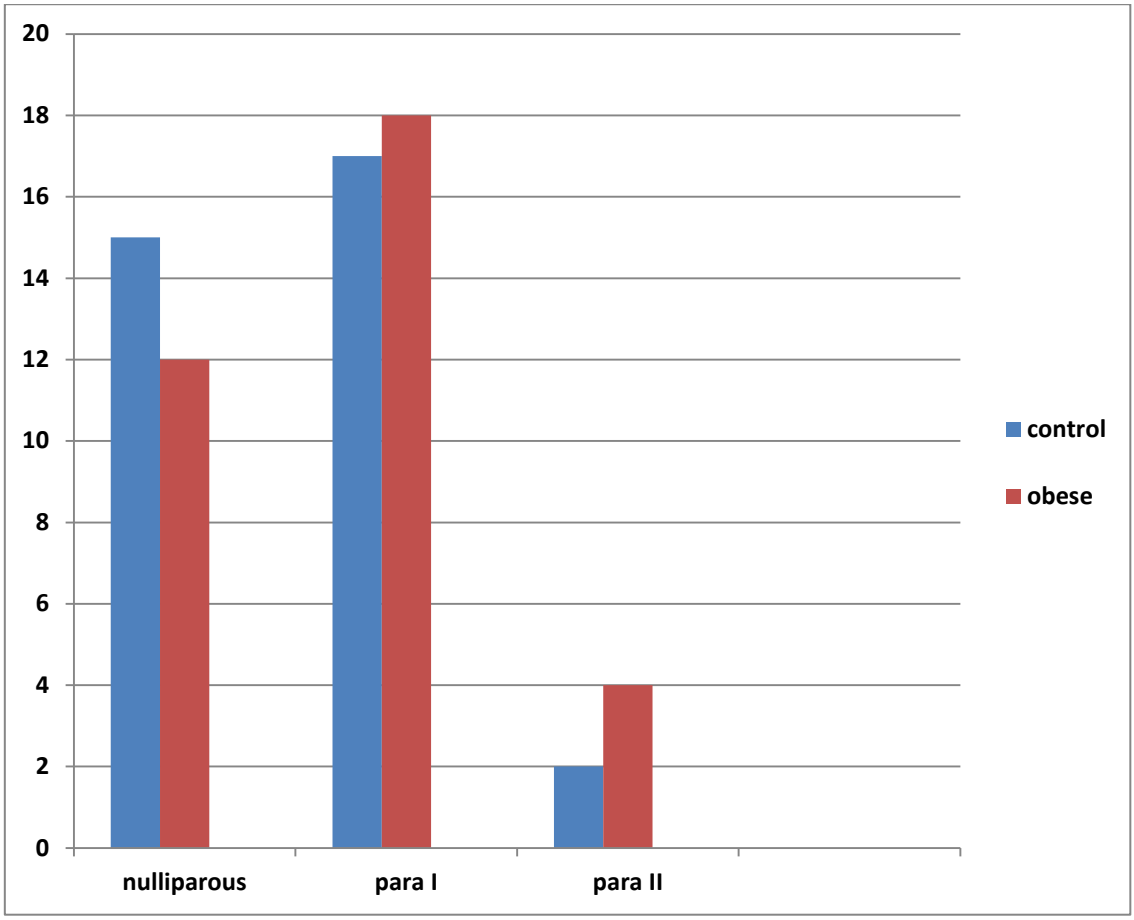


CATEGORISATION OF OBESE WOMEN

BMI kg/m2	Category	Numbers	Percentage
30-34.9	Moderate obesity	28	82.35%
35-39.9	Severe obesity	4	11.76%
>40	Very severe obesity	2	5.88%

In the study group 82.35% were moderately obese,11.76% were severely obese and only 5.88% were very severely obese.

PARITY


20
18
16
14
12
10
8
6
4
2
0
nulliparous
para I
para II
control
obese

PARITY

Parity	Control		Obese	
	No	Percentage	No	Percentage
Nulliparous	15	44.11%	12	35.29%
Para I	17	50%	18	52.94%
Para II	2	5.88%	4	11.76%

X2=11.02,P=0.02(significant)

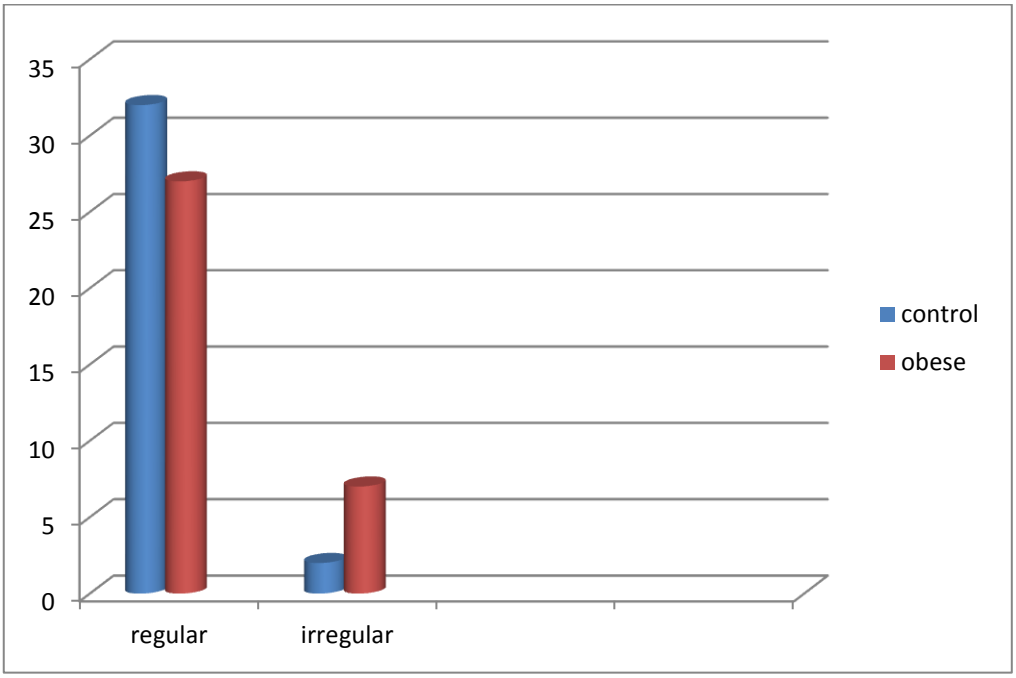
Among obese women 35.29% were nulliparous and 64.71% were parous women, where as in control group 44.11% were nulliparous and 55.89% were parous women.

MEAN BMI IN OBESE POPULATION IN RELATION TO PARITY

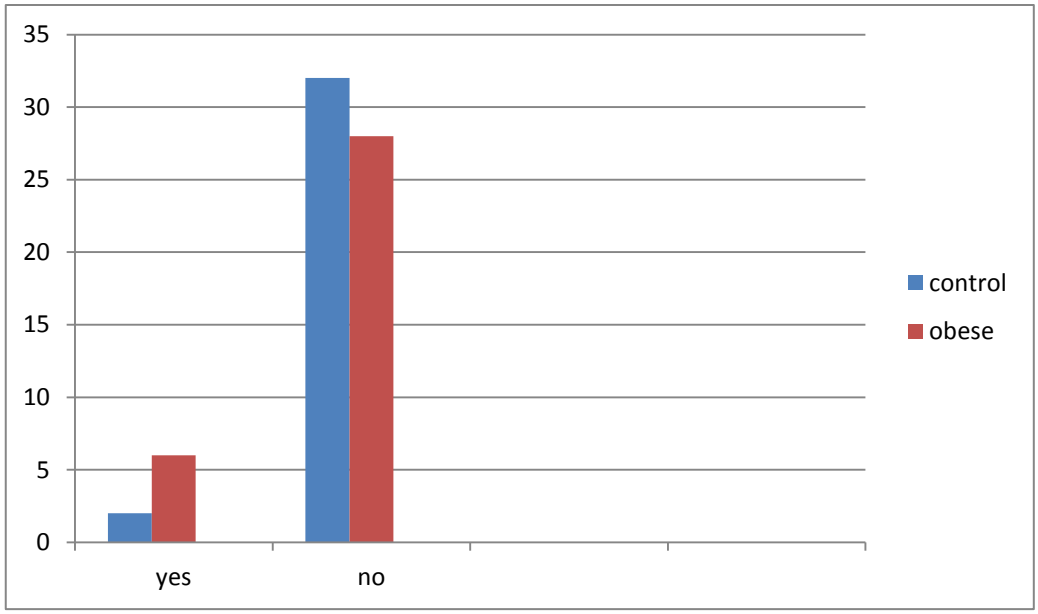
Parity	Mean BMI(kg/m2)
Nulliparous	32.09
Para I	32.87
Para II	34.38

As parity increased the mean BMI increased among obese women.

MENSTRUAL PATTERN



INFERTILITY



MENTRUAL PATTERN

Menstrual pattern	Control		Obese	
	No	Percentage	No	Percentage
Regular	32	94.11%	27	79.41%
Irregular	2	5.88%	7	20.58%

X2=22.7,P=0.001(significant)

20.58% of obese women had irregular menstrual pattern where as only 5.88% of control women had irregular menstrual pattern.

INFERTILITY

Infertility	Control		Obese	
	No	Percentage	No	Percentage
Yes	2	5.88%	6	17.64%
No	32	94.11%	28	82.35%

X2=27.3,P=0.001(significant)

In obese women 17.64% had infertility whereas in control women it was 5.88%

PREPREGNANCY MEDICAL DISORDERS

Medical disorders	Control		Obese	
	No	Percentage	No	Percentage
Diabetes	0	-	1	2.94%
Hypertension	0	-	1	2.94%
Hypothyroidism	1	2.94%	4	11.76%
Asthma	0	-	1	2.94%
Epilepsy	1	2.94%	1	2.94%
Heart disease	1	2.94%	0	-

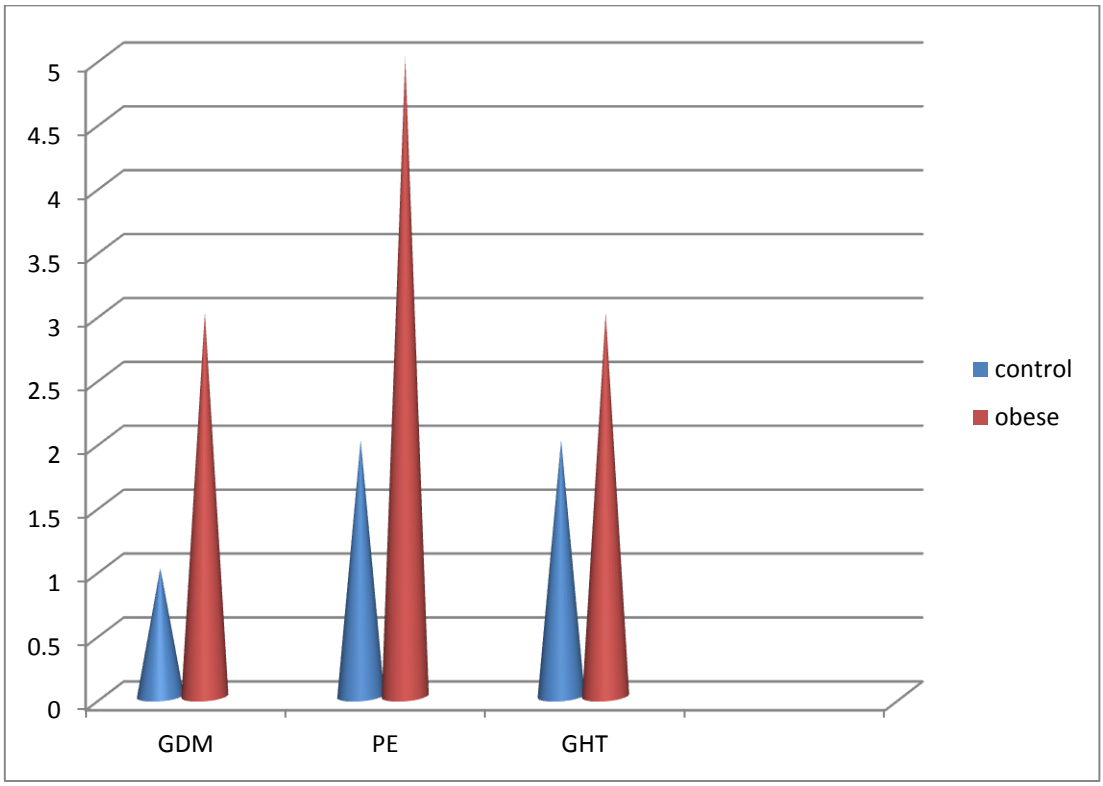
One were diabetic in obese group, where as none were so in control group.

One were hypertensive in obese group, where as none were so in control group.

These were not statistically significant as were other disorders namely asthma, epilepsy and heart disease.

4 obese women (11.76%) were hypothyroid but in control group one were so (2.94%). This difference was statistically significant (P=0.001)

PREGNANCY RELATED MEDICAL DISORDERS



PREGNANCY RELATED MEDICAL DISORDERS

Complications	Control		Obese	
	No	Percentage	No	Percentage
Gestational diabetes mellitus	1	2.94%	3	8.82%
Pre eclampsia	2	5.88%	5	14.70%
Gestational hypertension	2	5.88%	3	8.82%

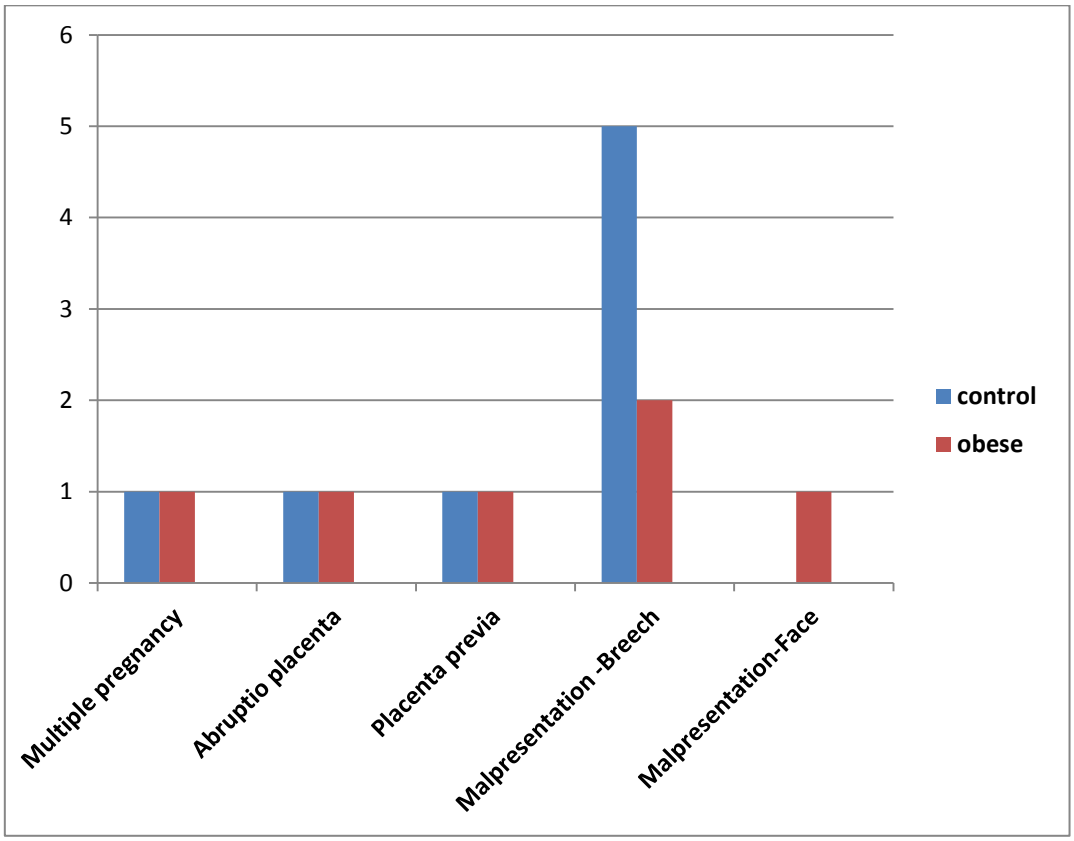
The incidence of gestational diabetes was 8.82% and 2.94% respectively in obese and control group.

The incidence of pre eclampsia was 14.70% and 5.88% in obese and control group.

The incidence of gestational hypertension was 8.82% and 5.88% in obese and control group.

The results were statistically significant.

OTHER OBSTETRICS COMPLICATIONS



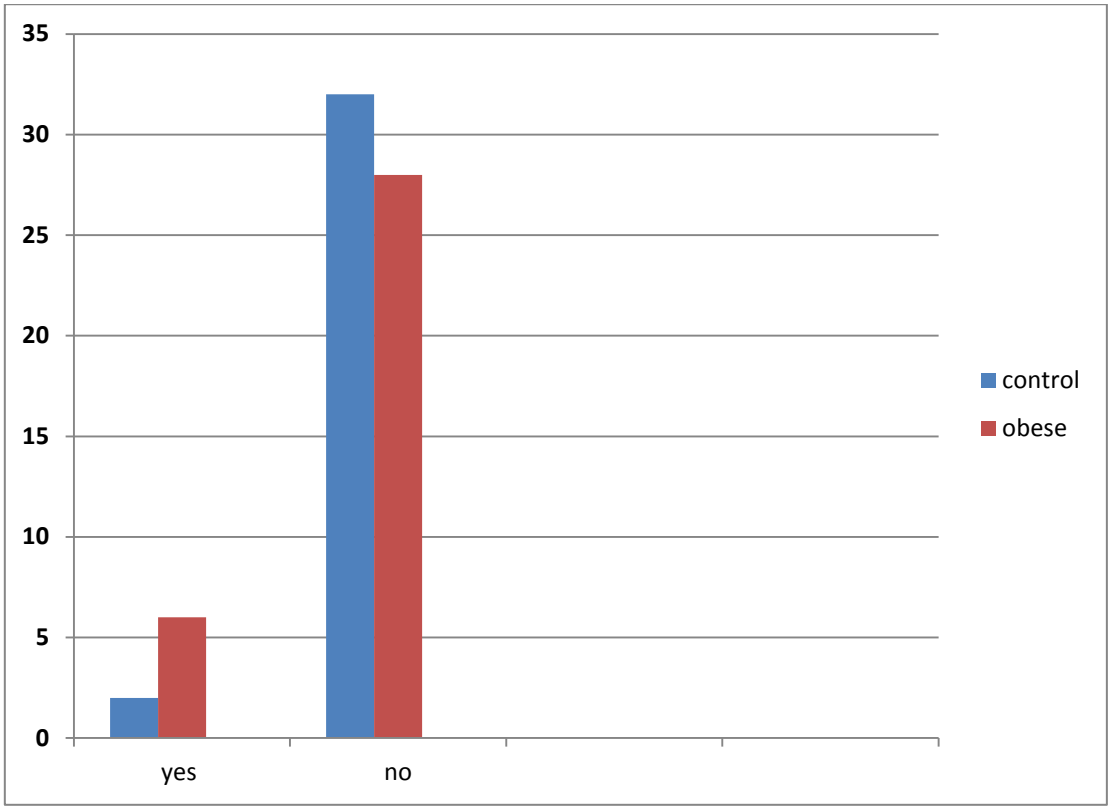
OTHER OBSTETRICS COMPLICATIONS

Complications	Control		Obese	
	No	Percentage	No	Percentage
Multiple pregnancy	1	2.94%	1	2.94%
Abruptio placenta	1	2.94%	1	2.94%
Placenta previa	1	2.94%	1	2.94%
Malpresentation				
Breech	5	14.70%	2	5.88%
Face	-	-	1	2.94%

P>0.05(not significant)

Obstetric complications like Multiple pregnancy, Placenta previa, Abruptio placenta and Malpresentation existed in both groups, but the difference was not statistically significant.

INDUCTION OF LABOUR



INDUCTION OF LABOUR

Induction	Control		obese	
	No	**Percentage**	**No**	**Percentage**
Yes	2	5.88%	6	17.64%
No	32	94.11%	28	82.35%

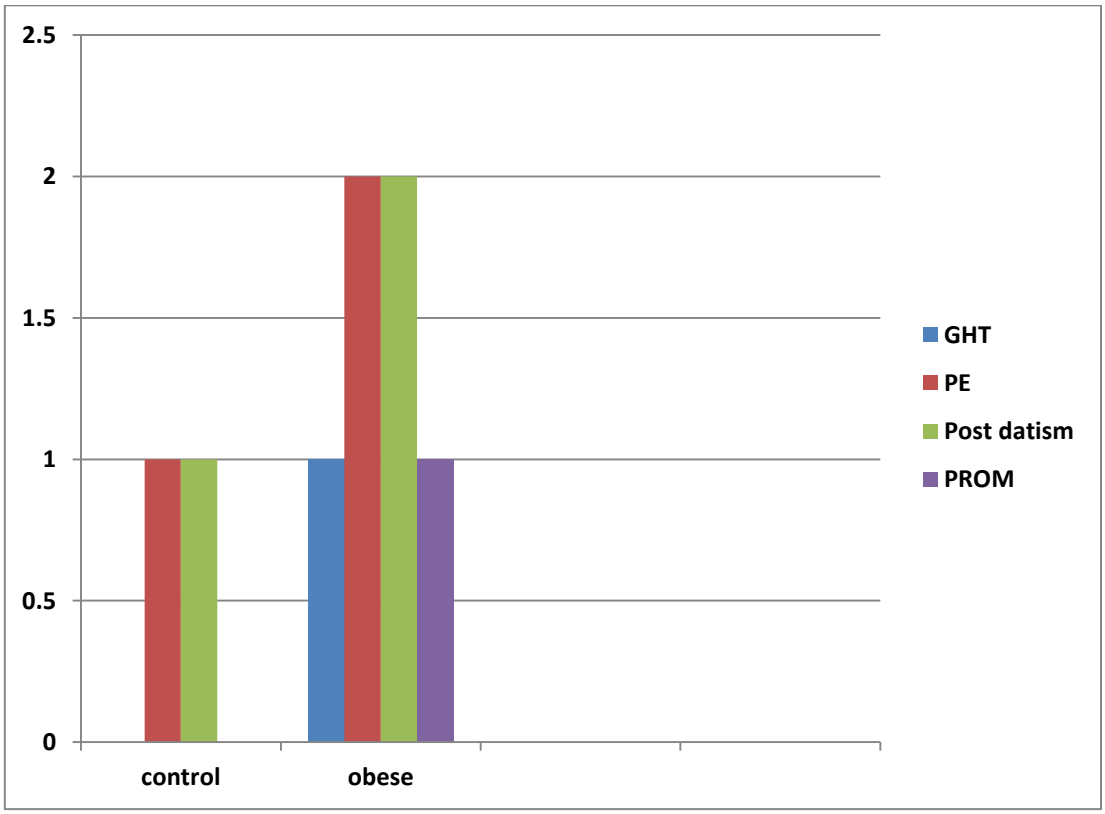
X2=3.84,P<0.05(significant),Odd's ratio=2.55

The labour induction rates were 17.64% and 5.88% in obese and control group respectively.

The rates were higher in obese group and the difference was statistically significant.

Obese women had 2.5 times increased risk of being induced than control women.

INDICATIONS FOR LABOUR INDUCTION

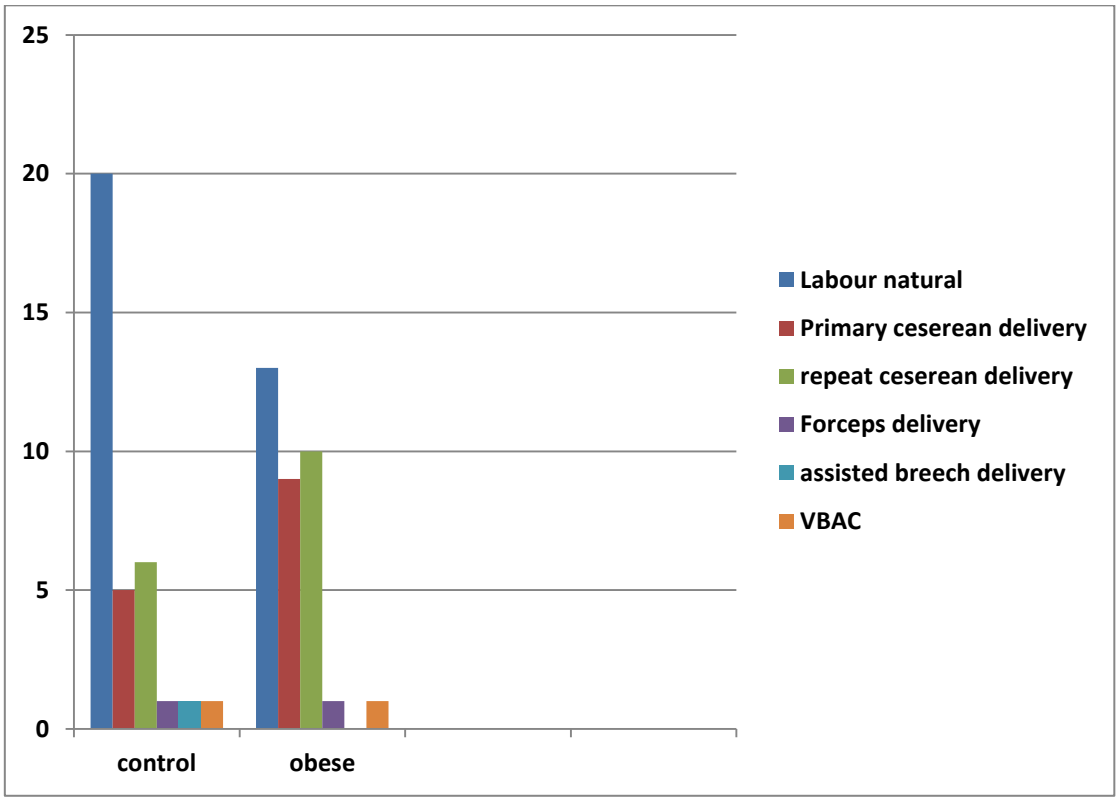


INDICATIONS FOR LABOUR INDUCTION

Indications	Control		Obese	
	No	Percentage	No	Percentage
Gestational hypertension	-	-	1	16.66%
Pre eclampsia	1	50%	2	33.33%
Post datism	1	50%	2	33.33%
PROM	-		1	16.66%

In both obese and control group, majority of induction of labour was done for Pre eclampsia and post datism.

MODE OF DELIVERY



MODE OF DELIVERY

Mode of delivery	Control		Obese	
	No	Percentage	No	Percentage
labour natural	20	58.82%	13	38.23%
Primary cesarean delivery	5	14.70%	9	26.47%
Repeat cesarean delivery	6	17.64%	10	29.41%
Forceps delivery	1	2.94%	1	2.94%
Assisted breech delivery	1	2.94%	0	0
VBAC	1	2.94%	1	2.94%

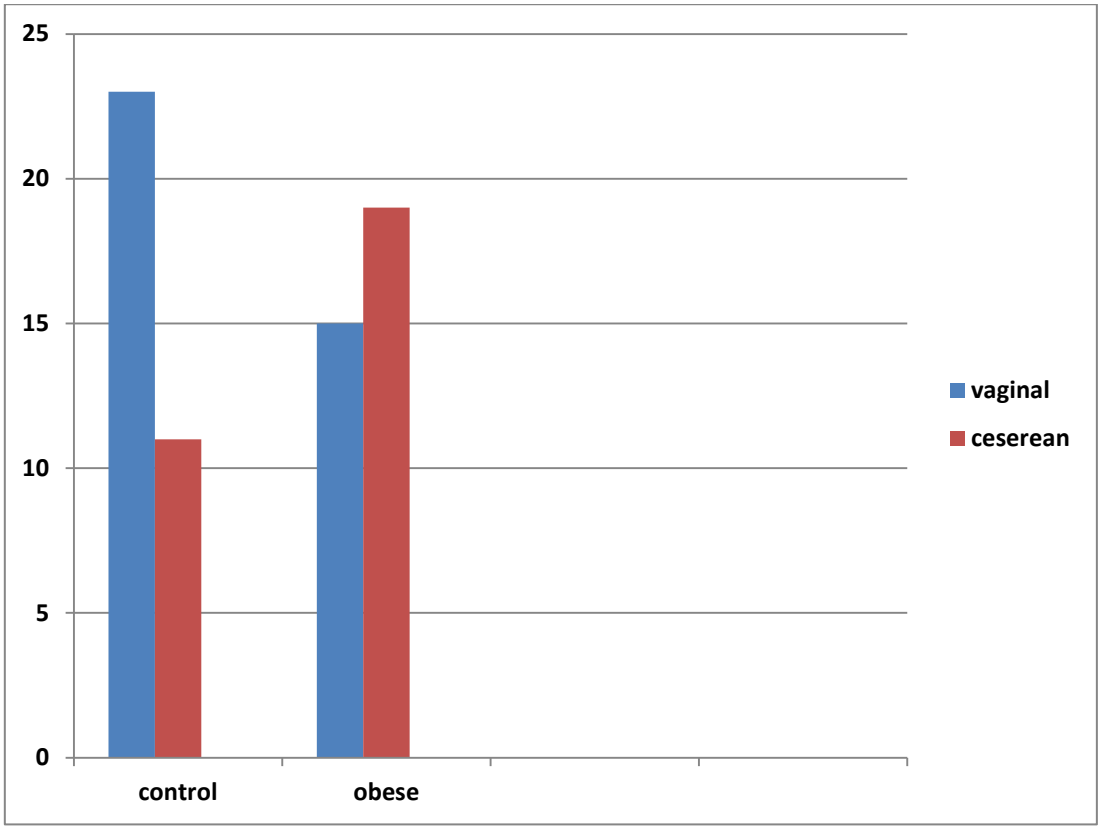
X2=19.51,P=0.001(significant)

The labour natural was lower in obese group(38.23%) when compared to control group(58.82%).

The primary cesarean delivery rates were higher in obese group(26.47%) when compared to control group(14.70%)

The instrumental delivery rates and VBAC rates were equal in both groups.

CESEREAN DELIVERY RATES



CESEREAN DELIVERY RATES

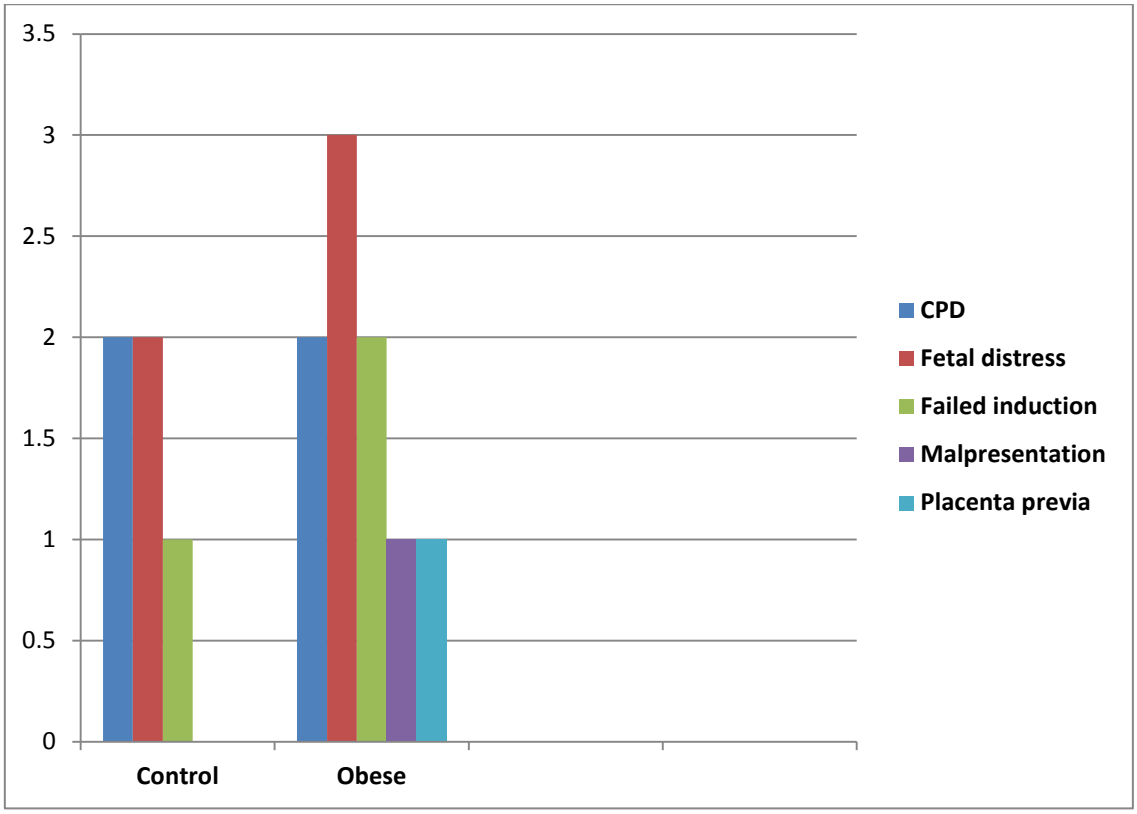
Mode of delivery	Control	Obese			
		moderate	severe	Very severe	Total
Vaginal	23(67.64%)	13(48.14%)	1(33.33%)	1(25%)	15(44.11%)
Cesarean	11(32.35%)	14(51.85%)	2(66.66%)	3(75%)	19(55.88%)

P=0.001(significant)

The cesarean delivery rates were higher in obese group(55.88%) than control group(32.35%).

The risk increased with severity of obesity.

INDICATIONS FOR PRIMARY CESEREAN DELIVERY



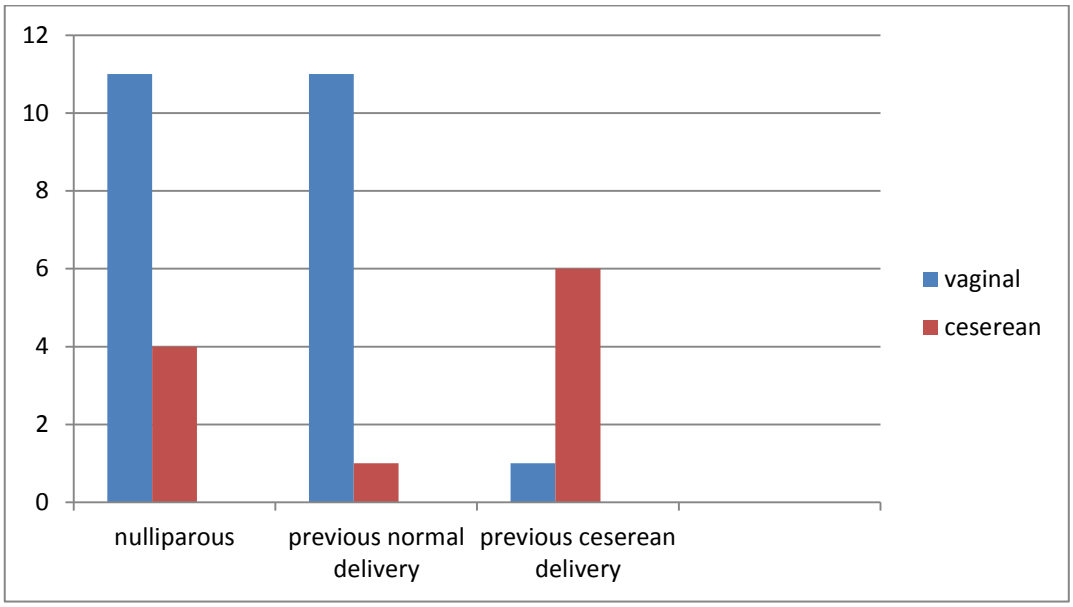
INDICATIONS FOR PRIMARY CESEREAN DELIVERY

Indications	Control		Obese	
	No	Percentage	No	Percentage
CPD	2	40%	2	22.22%
Fetal distress	2	40%	3	33.33%
Failed induction	1	20%	2	22.22%
Malpresentation	0	0	1	11.11%
Placenta previa	0	0	1	11.11%

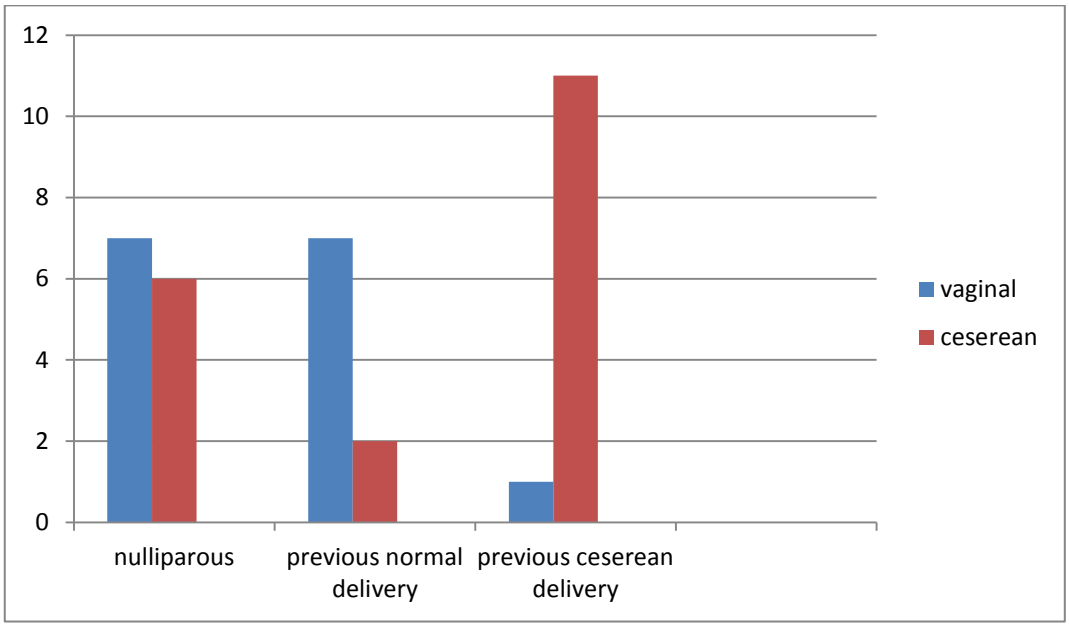
The major reasons for primary cesarean delivery were Fetal distress, CPD and failed induction in both groups.

MODE OF DELIVERY ACCORDING TO PARITY

CONTROL



OBESE



MODE OF DELIVERY ACCORDING TO PARITY

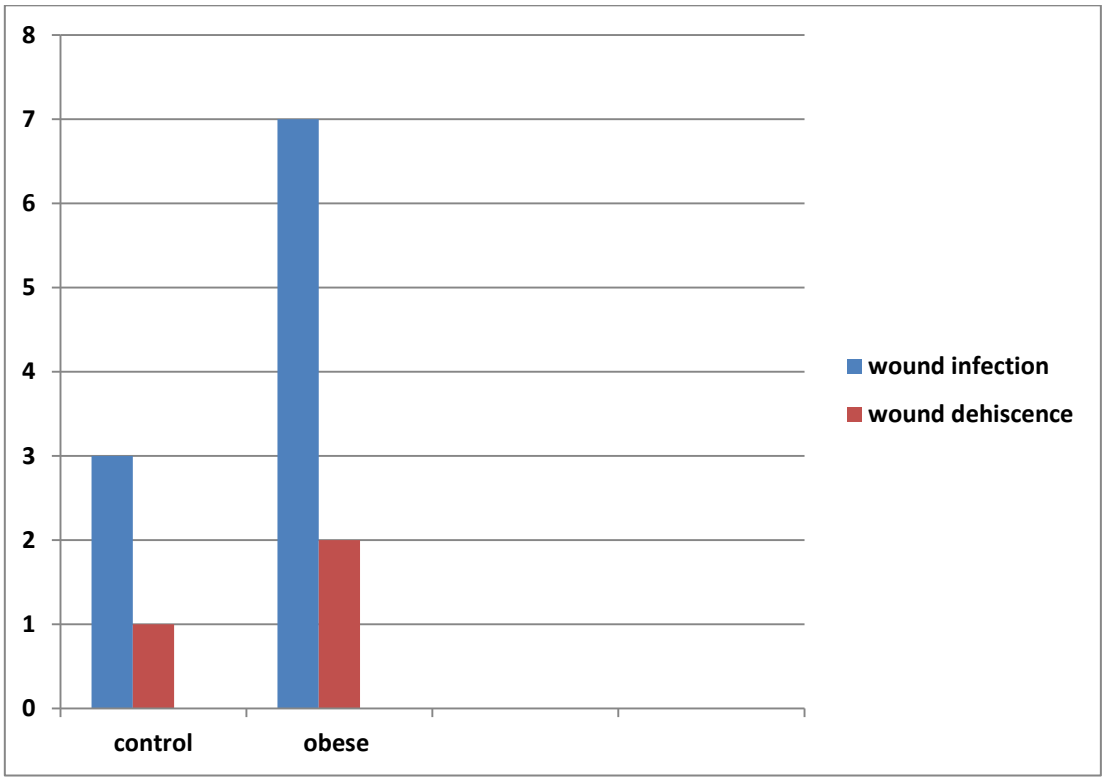
Mode of delivery	Control			Obese		
	Nulliparous (15)	**Previous normal delivery (12)**	**Previous cesarean delivery (7)**	**Nulliparous (13)**	**Previous normal delivery (9)**	**Previous cesarean delivery (12)**
Vaginal	11(73.33%)	11(91.66%)	1(14.28%)	7(53.84%)	7(77.77%)	1(8.33%)
Cesarean	4(26.66%)	1(8.33%)	6(85.71%)	6(46.15%)	2(22.22%)	11(91.66%)

In nulliparous women, cesarean delivery was higher in obese group(46.15%) when compared to control group(26.66%).(P=0.01 significant)

Similarly in parous women with previous normal delivery, cesarean delivery was higher in obese group (22.22%) than control group(8.33%) (P=0.01 significant).

The repeat cesarean rate was almost similar in both groups.

POSTPARTUM COMPLICATIONS



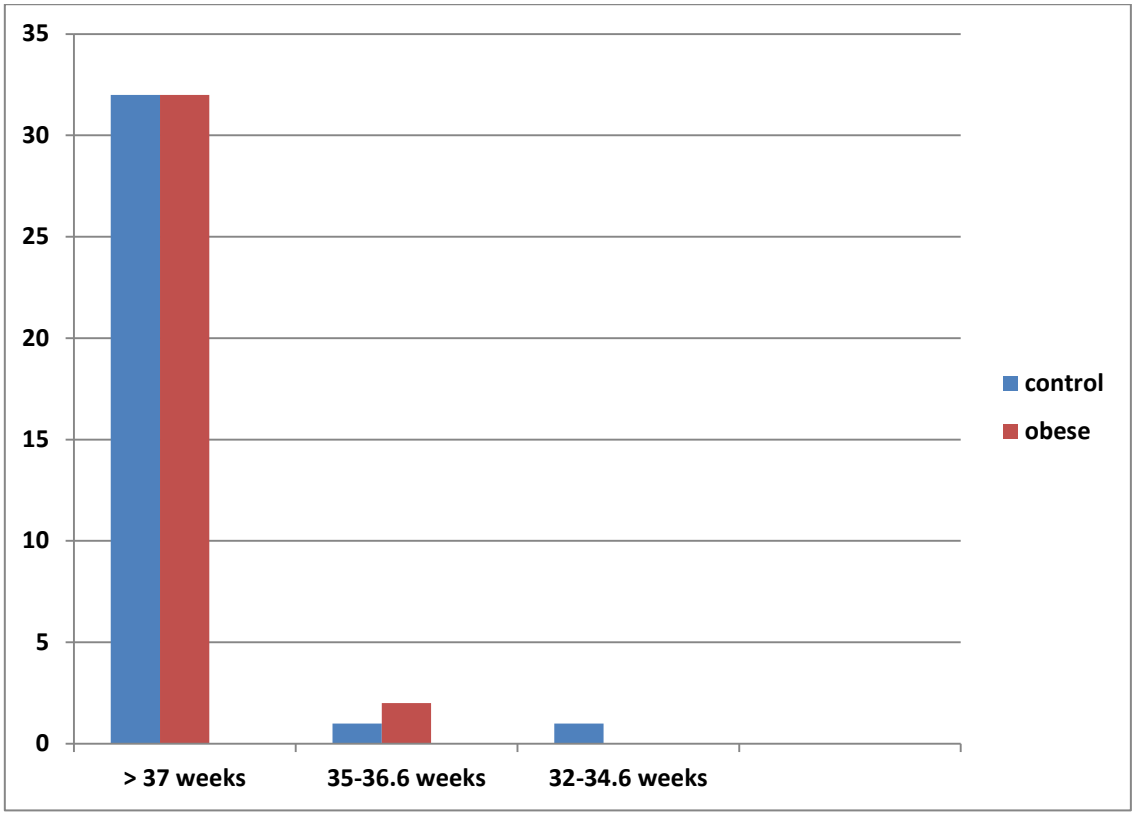
POSTPARTUM COMPLICATIONS

Complications	Control		Obese	
	No	Percentage	No	Percentage
Wound infection	3	8.82%	7	20.58%
Wound dehiscence	1	2.94%	2	5.88%

P <0.05 significant

Wound infection and dehiscence rates were higher in obese group (20.58% and 5.88%) than control group (8.82% and 2.94%).

GESTATIONAL AGE AT DELIVERY



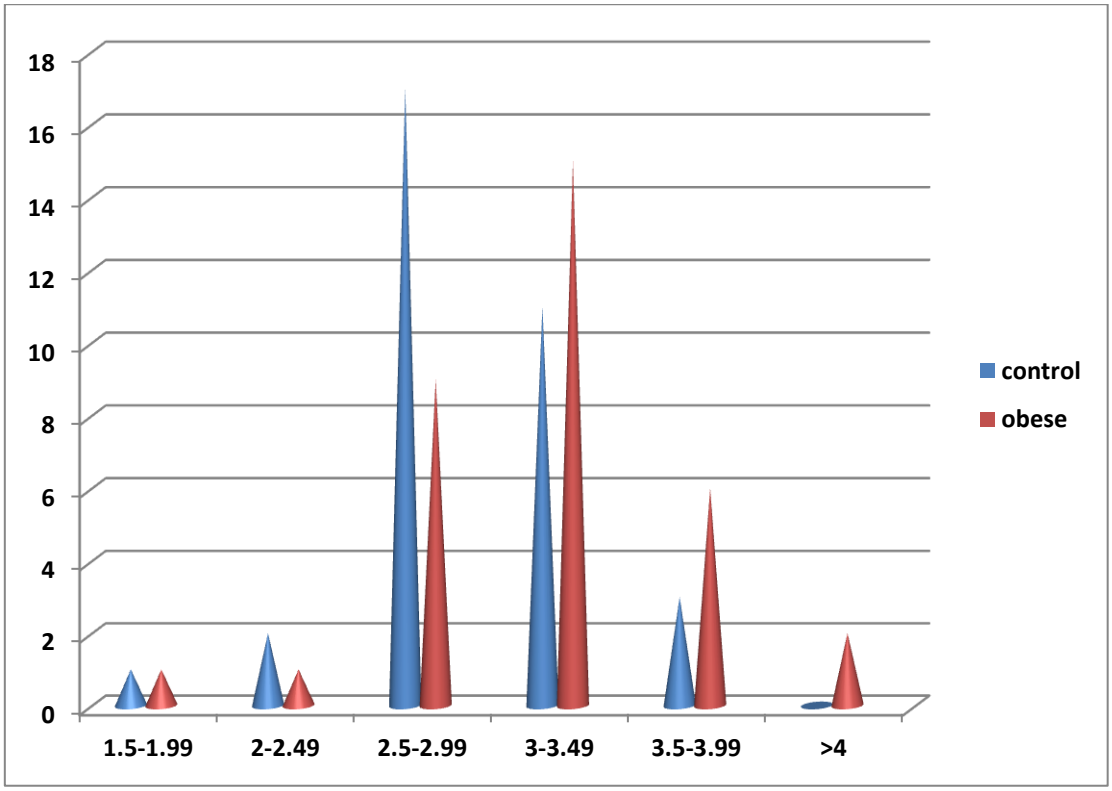
GESTATIONAL AGE AT DELIVERY

Gestational age (weeks)	Control		Obese	
	No	Percentage	No	Percentage
>37	32	94.11%	32	94.11%
35-36.6	1	2.94%	2	5.88%
32-34.6	1	2.94%	0	0

X2=0.65 P=0.72 not significant

94.11% of obese women and 94.11% of control women delivered at term. 5.88% of obese women and 5.88% of control group delivered preterm. The difference was not statistically significant.

BIRTH WEIGHT OF THE NEONATES



BIRTH WEIGHT OF THE NEONATE

Birth weight(kg)	Control		Obese	
	No	Percentage	No	Percentage
1.5-1.99	1	2.94%	1	2.94%
2-2.49	2	5.88%	1	2.94%
2.5-2.99	17	50%	9	26.47%
3-3.49	11	32.35%	15	44.11%
3.5-3.99	3	8.82%	6	17.64%
>4	0	0	2	5.88%
Total	34		34	

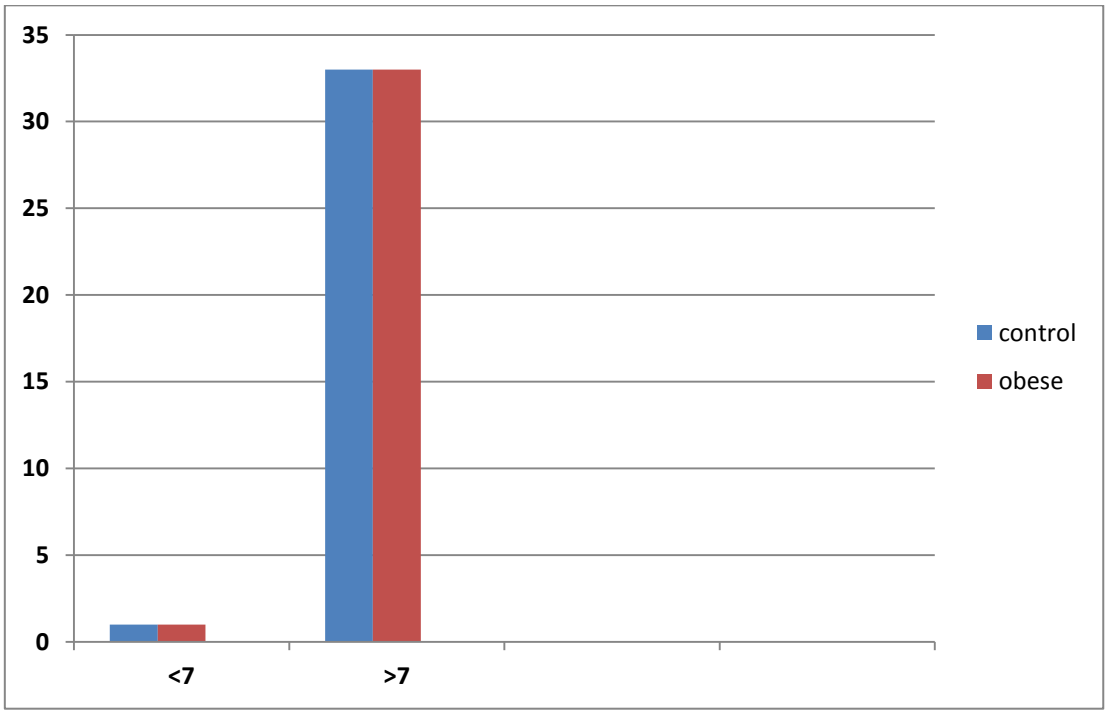
P<0.05 significant

Majority of the neonates of obese women(44.11%) were between 3-3.49 kg and of control women (50%) were between 2.5 – 2.99 kg.

17.64% babies of obese women were between 3.5-3.99 kg when compared to 8.82% babies of control women.

2 babies were > 4kg in obese women but none in control group.

APGAR AT 5 MINUTES



MEAN BIRTH WEIGHT OF NEONATE

	Numbers	Mean(kg)	Standard deviation	Student t-test
Control	34	2.92	0.323	T=4.80
obese	34	3.16	0.442	P=0.001

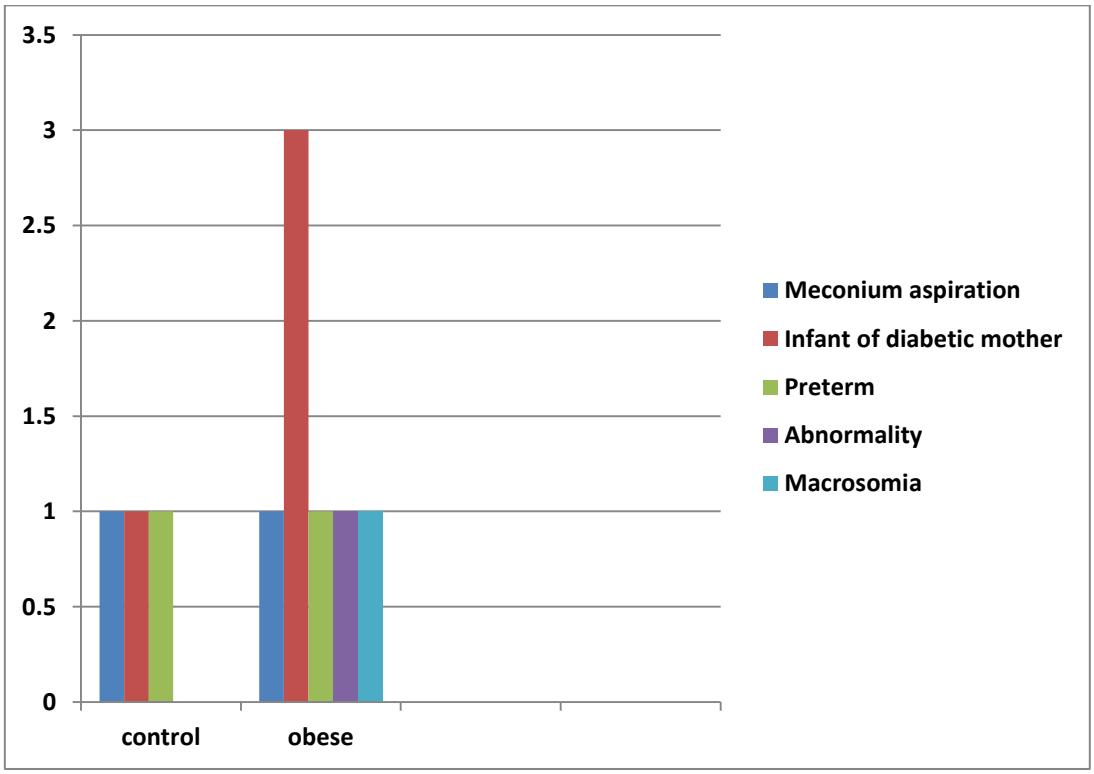
The mean birth weight of neonate was 3.16 kg in obese group and 2.92 kg in control group.

APGAR AT 5 MINUTES

Apgar at 5 minutes	Control		Obese	
	No	Percentage	No	Percentage
<7	1	2.94%	1	2.94%
≥7	33	97.06%	33	97.06%

The difference of APGAR at 5 minutes between obese and control group was not statistically significant (P>0.05).

INDICATION FOR NICU ADMISSION

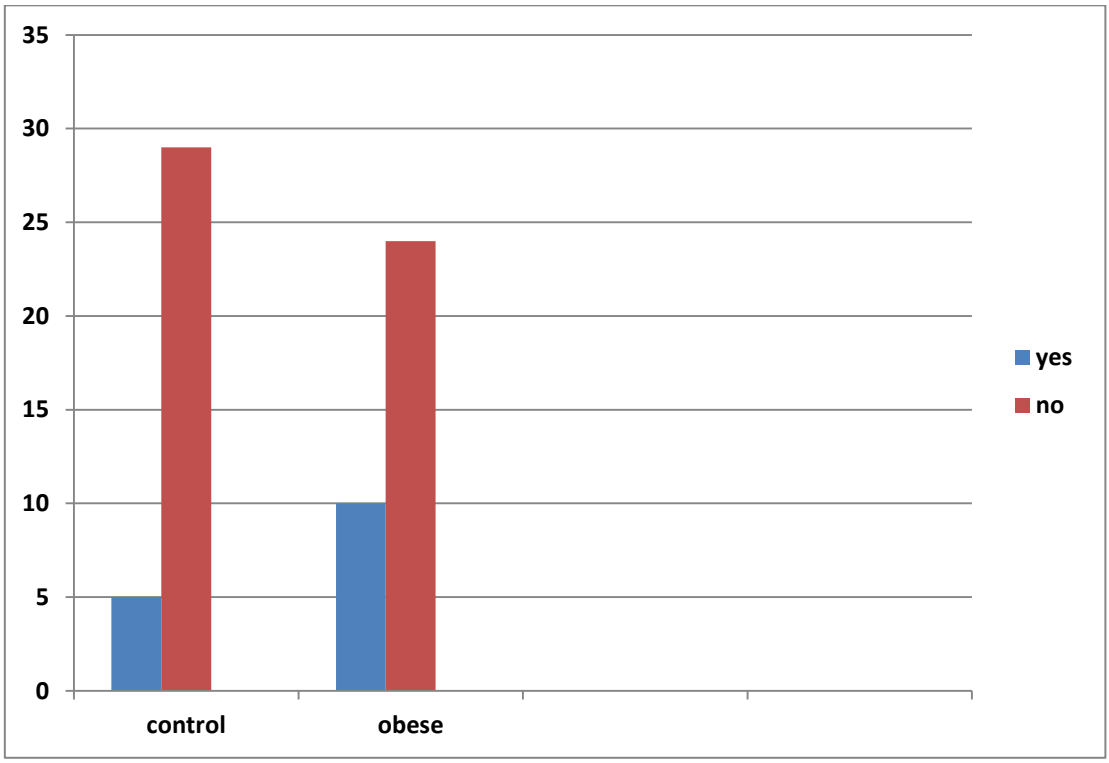


NICU ADMISSIONS AND THEIR INDICATIONS

Indications	Control		Obese	
	No	**Percentage**	**No**	**Percentage**
Meconium aspiration	1	33.33%	1	14.28%
Infant of diabetic mother	1	33.33%	3	42.85%
Preterm	1	33.33%	1	14.28%
Abnormality	0	0	1	14.28%
Macrosomia	0	0	1	14.28%
Total	3	8.82%	7	20.58%

20.58% of babies born to obese women and 8.82% of babies born to control were admitted in NICU ($P<0.05$)

PROLONGED HOSPITAL STAY



HOSPITAL STAY

	Hospital stay	Control		Obese	
		No	percentage	No	Percentage
Vaginal delivery	2 days	20	86.95%	11	73.33%
	>2 days	3	13.05%	4	26.66%
Cesarean delivery	7 days	9	81.81%	13	68.42%
	>7 days	2	18.18%	6	31.57%

P<0.05 significant

Among vaginal delivery group 26.66% of obese women and 13.05% of control women required prolonged hospital stay (>2 days).

Among cesarean delivery group 31.57% of obese women and 18.18% of control women required prolonged hospital stay (>7 days).

DISCUSSION

In our study, women in the obese group were slightly older when compared to women with normal BMI. The mean maternal age in obese group was 27.01 yrs. Obese women were less likely to be nulliparous. Mean BMI in obese group increased with parity. This is in accordance with the results of Ehrenberg HM et al 2002 that increasing age and parity are risk factors for obesity.

We observed that obese women had increased menstrual abnormalities and infertility when compared to women with normal BMI. This is consistent with studies done by Hartz AZ et al 1979 and Neil and Nelson 2001 that, obese women have menstrual abnormalities related to ovulatory dysfunction and insulin resistance leading to infertility.

Previous studies show that obese women have increased incidence of preexisting diabetes and chronic hypertension complicating pregnancy (Perlow et al 1992,Garbaciak 1985).But our study failed to show such association, which may be due to the small size of the sample. Obese women had increased incidence of Hypothyroidism (11.76%) in accordance with Garbaciak et al 1985.

In obese group, we found increased risk of Pre-eclampsia (14.70%).Obese women were observed to have an increased incidence of Gestational hypertension(8.82%) when compared with control group (5.88%).obese women observed to have higher risk of developing gestational diabetes (8.82%) when compared to normal BMI group(2.94%).

In our study placental abnormalities such as placenta previa and placental abruption occurred equally among obese women and normal weight women. Bainco et al showed an increased incidence of abruption but results of Wolf HM et al 1994 including ours did not show association.

There was no significant association with multiple pregnancy and BMI in our study, which occurred equally in obese group (2.94%) and control group(2.94%).This is consistent with study done by Marie.I Cedergren. But other studies have reported increased incidence of multiple pregnancy.(Gross T et al 1980,Naeye RL,1990)

We observed that labour induction was more common in obese group (17.64%) when compared to control group(5.88%) which is in accordance with other studies (Ekblad U et al 1992).The risk of induction among the obese women was increased almost 2.5 fold. Cedergren et al

2004 in his study had an incidence ranging fro 13.1% to 18.3% according to the severity of obesity. In our study the major reason for the induction was hypertensive disorders of pregnancy(50%) in obese group.

In the obese group, our results supported a number of previous studies(Joshua L Weiss et al 2001 and Marie I Cedergren 2004) that have demonstrated an increased risk for cesarean delivery in this group.

The cesarean delivery rates were 55.88% in obese group and 32.35% in control group. Obese women had 2.8 fold increased risk of cesarean delivery when compared to control group. The risk increased with the severity of obesity. The primary cesarean delivery rates were higher among obese group (26.47%) when compared to the control group (14.70%). The cesarean delivery rates were higher among nulliparous obese group and even obese women with previous normal delivery had higher risk of cesarean delivery. We also found that no difference in repeat cesarean delivery rates between both groups.

Instrumental deliveries were surprisingly not increased in obese group, which is in contrast to other studies (Joshua L Weiss et al 2001, Marie I Cedergren 2004).The increased cesarean delivery rates in obese women may explain why we did not find association between instrumental delivery and obesity. But in a large study from London

(Sebire NJ, et al 2001) no increased risk of instrumental delivery was seen among obese women. Complete perineal tear and shoulder dystocia was not seen in either groups, which may be due to increased cesarean delivery rates and low instrumental delivery rates.

In accordance with other studies (Myles et al 2002, Wolf HM et al 1998)we found obese women to be at a greater risk of post operative wound infection and wound dehiscence.

There are conflicting data in the litherature regarding maternal obesity and preterm birth, with some studies (Baeten et al 2001) showing increased risk and some studies showing no change (Sebire et al 2001).In our study no difference was found between either groups for preterm birth < 37 weeks. The reason for the difference in study results may reflect difference in study population.

In our study, the mean birth weight of the neonates of obese women was 3.16 kg and the neonates of control group was 2.92 kg. As previously reported, (Ehrenberg et al, Sebire et al 2001) obese women had increased risk of delivering high birth weight babies. We found that 23.52% of obese group delivered babies 3.5 kg and above, when compared to 8.82% of control group.

Neonates of obese mothers had increased NICU admission, the major reasons for admission being Infants of diabetic mother. There was no difference in APGAR score at 5 min between the two groups. This is consistent with study done by Line Rode et al.

As documented in previous studies, (Hood et al 1993)the obese women had prolonged hospital stay, which may be due to associated medical complications, wound infection and NICU admission.

SUMMARY

In our study,34 obese women(BMI>30 kg/m2) and 34 women with normal BMI(18.5 kg/m2 to 24.99 kg/m2) were studied.It was observed that

1. Obese women were slightly older than control group. Majority of obese women belonged to age group 25-29 yrs when compared to control group who belonged to 20-24 yrs age group.

2. The mean age of obese women was 27.01 yrs and that of control women was 24.14 yrs.

3. The proportion of nulliparous women was less in obese group (35.29%)when compared to control group (44.11%)

4. In obese group, the mean BMI increased with increase in parity.

5. Among obese group, majority (82.35%)were moderately obese,11.76% were severely obese and 5.88% were very severely obese.

6. 20.58% of obese women had menstrual abnormalities when compared to 5.88% of control women.

7. Infertility was seen in 17.64% of obese group and 5.88% in control group.

8. Obese women had increased incidence of pre-existing medical disorders like hypothyroidism when compared to control group. But no difference was seen with respect to diabetes, hypertension and other morbidities between the two groups.

9. Obese women had increased incidence of gestational diabetes when compared to control group(8.82% vs 2.94%).

10. The incidence of pre-eclampsia was higher in obese group when compared to control group(14.70% vs 5.88%)

11. Gestational hypertension was found to be higher in obese group when compared to control group(8.82% vs 5.88%)

12. Obese women were more likely to be induced (17.64%,Odd's ratio:2.5)when compared to control group(5.88%)

13. Increased cesarean delivery rates was found among obese women (55.88%)when compared to control group(32.35%).The risk increased with increase in severity of obesity.

14. Nulliparous women had 2.5 fold increased risk of cesarean delivery in obese group when compared to women with normal BMI.

15. No difference was seen among obese and control group with respect to placenta previa, abruptio placenta, multiple pregnancy, instrumental deliveries, shoulder dystocia and complete perineal tears.

16. Post operative wound infections and wound dehiscence were found to be increased in obese group(20.58%,5.88%)when compared to control group(8.82%,2.94%) respectively.

17. No difference was found in preterm births (<37 weeks) between two groups.

18. The majority of the neonates of obese women (44.11%) were between 3kg-3.49 kg where as majority of neonates in control group (50%) were between 2.5kg-2.99kg.

19. Two babies of obese women were >4kg but none were in control group.

20. No difference was seen among obese and control group with respect to APGAR score at 5 minutes.

21. There were increased admissions to NICU among neonates of obese women (20.58%) when compared to control group (8.82%). The major reasons for admissions were for the care of infant of diabetic mother.

22. Prolonged hospital stay was required in obese group (29.41%) when compared to control group(14.70%).The major reasons for the prolonged stay were due to wound infections, medical disorders and NICU admissions.

CONCLUSION

Our study points out the numerous maternal and perinatal risks in obese pregnant women which pose a considerable challenge to the obstetrical practitioner. In addition, massive obesity among women of child bearing age is associated with a number of health risks later in life. This stresses the importance of concentrating on trying to reduce the increasing incidence of obesity in fertile women. The best time of intervention may be before a women considers a pregnancy, because it is not recommended that obese women lose weight during pregnancy.

This implicates the need of pre-pregnancy advice and counseling to young women. Obese women considering pregnancy should be informed of the risk that maternal obesity confers on a pregnancy.

Health care professionals need to encourage and assist obese women to make life style changes, to lose weight pre-conceptually in an attempt to optimize and potentially decrease the risk of complication in pregnancy.

Pregnancies among obese women must be classified as high risk pregnancies and appropriate care should be provided with heightened surveillance, anticipation and diagnosis of the complications and intervene earlier if complications arise.

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PROFORMA

NAME :

AGE :

IP NO :

PHONE NUMBER :

ADDRESS :

HUSBANDS NAME :

OCCUPATION :

QUALIFICATION :

SOCIOECONOMIC STATUS :

BOOKING :

IMMUNISATION :

H/O PRESENT ILLNESS :

MENSTRUAL HISTORY : regular/irregular

LMP :

EDD :

MARITAL HISTORY : Married since

Consangunity :

h/o infertility :

OBSTETRIC HISTORY :G P L A

Last child birth

PREVIOUS OBSTRETIC HISTORY: details of outcome

PAST MEDICAL HISTORY

1. Diabetes
2. Hypertension
3. Heart disease
4. Others
5.drug intake
6.childhood obesity
7.Hypothyroidism
8.Renal disorders.

PAST SURGICAL HISTORY:

PERSONAL HISTORY:

PRESENT PREGNANCY:

I Trimester:

Hyperemesis
Fever
Radiation exposure
Medications
Pain abdomen

II Trimester:

Date of quickening
Bleeding P/V
GDM
Pre-eclampsia

III Trimester:

Bleeding P/V
GDM
Pre-eclampsia

GENERAL EXAMINATION

HEIGHT AT BOOKING :

WEIGHT AT BOOKING:

BMI AT BOOKING:

RATE OF WEIGHT GAIN: II trimester
III trimester

TOTAL WEIGHT GAIN : >10kg
>12 kg
>20 kg

WEIGHT AT DELIVERY:

ANEMIA :

PEDAL EDEMA :

VITALS :

TEMP :

PULSE :

BP :

THYROID :

BREAST :

CVS :

RS :

SPINE :

GAIT :

OBSTRETIC EXAMINATION:

PER ABDOMEN :

Fundal height:
Abdominal girth;
Fundal grip:
Umblical grip:
First pelvic grip:
Second pelvic grip:
Fetal heart:
Liquor volume:
Estimated fetal weight:

PELVIC EXAMINATION :

Investigations

Urine-albumin
Sugar
C/S

Blood-Hb
PCV

OGCT

Urea
Creatinine

VDRL
HIV
HbsAg

TFT

Blood G&T

Lipid profile

USG:

ANTEPARTUM COMPLICATIONS:

Gestational diabetes
Pre-eclampsia
Gestational hypertension
Placenta previa
Abruptio placenta
Malpresentation
Hydromnios
Macrosomia

DELIVERY DETAILS:

Induction of labour : yes/no
Indication for induction :
Date of delivery :

MODE OF DELIVERY:

Labour natural VBAC
Forceps delivery
Cesarean delivery elective/emergency
Indication for cesarean delivery
Placenta weight
Baby weight

INTRAPARTUM COMPLICATIONS:

Shoulder dystocia
PPH
Complete perineal tear
Colour of liquor

POSTPARTUM COMPLICATIONS:

Wound infections
Wound dehiscence
Deep vein thrombosis
Fever
Lactational problems
Subinvolution of uterus

NEONATE:

Live born: still born: IUD:
APGAR 1min 5 min
Gestational age at delivery:
Birth weight:
Sex of the baby M: F:
Congenital abnormalities:
Admission in NICU:
Reason for admission in NICU:
Neonatal death:
Condition at discharge:
Date of discharge:

DURATION OF HOSPITAL STAY:

Vaginal delivery	2 days	>2 days
Cesarean delivery	7 days	>7 days

Signature of the Investigator **Signature of the Guide**

DATE: **DATE :**

“MATERNAL AND FETAL OUTCOME IN OBESITY COMPLICATING PREGNANCY-A PROSPECTIVE COHORT STUDY”

பங்குபெறுபவரின்பெயர் :

பங்குபெறுபவரின்வயது : **பங்குபெறுபவரின்எண் :**

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளல்லாம் என்றும் அறிந்துகொண்டேன்.

இந்த ஆய்வுசம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக்கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரி கையொப்பம் ஆய்வாளரின் கையொப்பம்

இடம் :

தேதி :

OBESE

s.no	Age	socio economic status	Height	Wt at booking	BMI at booking	Weight at delivery	Menstrual irregularity	Infertility	Obstetric index	Maternal complication	Labour induction	Indication for labour induction	Mode of delivery	Indication for Ceserean delivery	Postpartum complications	Gestational age at delivery	APGAR	Birth weight	NICU	Indication for admission	Hospital stay
1	30	IV	153	70	31	82	no	yes	primi	Placenta previa	no		primary CS	CPD		term	>7	2.5	no		7
2	25	V	153	65	34	77	no	no	primi	abruptio placenta	no		primary CS	placenta previa		term	>7	2.6	yes	meconium	7
3	20	V	147	61	32	73	yes	no	G3P2L2	GDM	yes	PROM	primary CS	CPD		term	>7	3	no		7
4	26	V	151	80	36	92	no	no	G2P1L1		no		repeat CS		WI	term	>7	3.6	no		8
5	31	III	152	66	30	78	no	no	G2P1L1	Pre eclampsia	no		repeat CS			term	>7	3.1	no		7
6	21	V	150	71	34	83	no	no	G2P1L1		yes	post datism	primary CS	fetal induction		36 weeks	<7	3.6	no		7
7	32	V	146	72	32	84	yes	no	G2P1L1	GDM	yes	post datism	repeat CS		WD	term	>7	1.7	yes	preterm	9
8	22	IV	145	67	30	79	no	yes	G3P2L2	GHT	no		repeat CS			term	>7	2.7	no		7
9	33	V	144	65	30	77	no	no	primi	GHT	no		primary CS	malpresentation		term	>7	2.8	no		7
10	19	II	151	64	32	76	yes	no	primi		no		primary CS	fetal distress		term	>7	3.9	yes	IDM	7
11	27	IV	154	80	36	90	no	no	primi	GDM	no		LN			term	>7	2.9	no		3
12	23	V	160	71	35	80	no	yes	primi		no		LN			36 weeks	>7	2.3	yes	abnormality	4
13	34	III	153	73	34	83	yes	no	G3P2L2	GHT	yes	GHT	repeat CS		WD	term	>7	4.1	yes	IDM	8
14	24	V	161	75	34	85	no	no	G2P1L1	Pre eclampsia	no		repeat CS		WI	term	>7	3.3	no		9
15	28	III	151	77	35	87	yes	no	G2P1L1	Pre eclampsia	no		repeat CS		WI	term	>7	3.5	no		7
16	24	IV	149	64	30	74	no	yes	G2P1L1		no		LN			term	>7	3.4	no		3
17	29	III	154	66	31	76	no	no	G3P2L2		no		repeat CS		WI	term	>7	3.3	no		8
18	33	V	148	68	32	78	yes	no	G2P1L1		no		repeat CS		WI	term	>7	2.9	no		9
19	23	IV	150	61	30	71	no	no	primi	Pre eclampsia	yes	Pre eclampsia	primary CS	fetal distress		term	>7	3.1	no		7
20	32	V	155	81	37	91	yes	no	G2P1L1		no		LN			term	>7	3.8	no		4
21	22	IV	154	85	38	95	no	no	primi	Pre eclampsia	yes	Pre eclampsia	primary CS	fetal induction		term	>7	2.8	no		7
22	31	V	156	70	34	80	no	yes	G2P1L1		no		LN			term	>7	3	no		2
23	21	V	156	74	34	84	no	yes	primi		no		LN			term	>7	2.7	no		2

s.no	Age	socio economic status	Height	Wt at booking	BMI at booking	Weight at delivery	Menstrual irregularity	Infertility	Obstetric index	Maternal complication	Labour induction	Indication for labour induction	Mode of delivery	Indication for Ceserean delivery	Postpartum complications	Gestational age at delivery	APGAR	Birth weight	NICU	Indication for admission	Hospital stay
24	29	V	157	76	33	86	no	no	G2P1L1		no		LN			term	>7	3.4	no		2
25	28	V	155	64	31	74	no	no	G2P1L1		no		repeat CS		WI	term	>7	2.6	no		7
26	27	IV	145	67	32	77	no	no	primi		no		primary CS	fetal distress	WI	term	>7	3.3	no		7
27	30	V	150	77	35	87	no	no	G2P1L1		no		VBAC			term	>7	4.4	yes	macrosomia	2
28	21	V	160	75	35	85	no	no	G2P1L1		no		LN			term	>7	3.3	no		2
29	25	IV	145	73	34	83	no	no	G2P1L1		no		LN			term	>7	3.1	no		2
30	26	IV	166	71	34	81	no	no	G2P1L1		no		LN			term	>7	3.4	no		2
31	27	IV	148	72	33	82	no	no	G2P1L1		no		forceps			term	>7	3	no		2
32	28	V	158	70	35	80	no	no	G2P1L1		no		LN			term	>7	3.4	yes	IDM	2
33	29	IV	155	68	32	80	no	no	primi		no		LN			term	>7	3.1	no		2
34	28	IV	154	64	32	76	no	no	primi		no		LN			term	>7	3.7	no		2

CONTROL																				
S.NO	AGE	Socio economic status	Height	Wt at booking	BMI at booking	Weight at delivery	Menstrual irregularity	Infertility	Obstetric index	Maternal complication	Labour induction	Indication for labour induction	Mode of delivery	Indication for Ceserean delivery	Postpartum complications	Gestational age at delivery	APGAR	Birth weight	NICU	Indication for admission	Hospital stay
1	20	IV	151	50	20	60	no	no	primi		no		LN			Term	>7	2.6	no		2
2	21	V	144	45	23	55	no	no	G2P1L1		no		Primary CS	fetal distress		Term	>7	2.5	no		7
3	19	V	145	41	21	51	no	no	G2P1L1	GDM	no		LN			Term	>7	3.3	no		2
4	22	IV	146	60	25	70	no	no	G3P2L2		no		LN			Term	>7	2.7	no		3
5	23	V	150	46	19	56	no	no	primi	Abruptio placenta	yes	pre eclampsia	LN			36 weeks	<7	2.6	no		4
6	18	IV	152	51	23	61	no	yes	G2P1L1		no		LN			Term	>7	3	no		5
7	24	V	151	52	21	62	no	no	G2P1L1	GHT	no		assisted breech			Term	>7	2.8	no		2
8	20	V	147	47	19	57	no	no	primi	Placenta previa	no		Repeat CS		WI	Term	>7	2.7	no		7
9	21	V	153	45	19	55	no	no	G2P1L1		no		LN			Term	>7	3.1	no		2
10	22	V	153	44	21	54	no	no	primi		no		LN			Term	>7	3.2	no		2
11	23	II	155	60	25	70	no	no	G2P1L1		no		Forceps			Term	>7	2.8	no		2
12	24	V	150	51	24	61	no	yes	G2P1L1		yes	post datism	LN			Term	>7	3	no		2
13	24	III	148	53	23	63	no	no	primi	GHT	no		LN			Term	>7	2.9	no		2
14	23	V	154	55	23	65	no	no	G2P1L1		no		LN			33 weeks	>7	3.6	no		2
15	22	V	149	57	24	67	no	no	primi		no		Repeat CS		WI	Term	>7	2.9	no		7
16	21	V	151	44	19	54	no	no	primi		no		Repeat CS		WI	Term	>7	2.8	no		7
17	20	IV	161	46	20	56	no	no	G2P1L1	Pre eclampsia	no		LN			Term	>7	2.7	no		2
18	21	III	153	48	21	58	no	no	G3P2L2		no		VBAC			Term	>7	3	no		2
19	22	V	160	41	19	51	no	no	G2P1L1		no		Primary CS	fetal distress		Term	>7	3.4	no		7

S.NO	AGE	Socio economic status	Height	Wt at booking	BMI at booking	Weight at delivery	Menstrual irregularity	Infertility	Obstetric index	Maternal complication	Labour induction	Indication for labour induction	Mode of delivery	Indication for Ceserean delivery	Postpartum complications	Gestational age at delivery	APGAR	Birth weight	NICU	Indication for admission	Hospital stay
20	23	IV	154	61	26	71	no	no	primi	Pre eclampsia	no		LN			Term	>7	2.5	no		2
21	24	V	155	65	27	75	no	no	primi		no		LN			Term	>7	3.7	no		2
22	25	V	155	50	23	60	no	no	primi		no		Repeat CS			Term	>7	2.9	no		9
23	29	V	155	54	23	64	no	no	G2P1L1		no		Repeat CS			Term	>7	2.6	no		8
24	28	V	150	56	22	66	no	no	G2P1L1		no		LN			Term	>7	2.9	no		2
25	26	IV	156	44	20	54	no	no	primi		no		Primary CS	fetal induction		Term	>7	2.1	yes	Preterm	7
26	30	V	156	47	21	57	no	no	G2P1L1		no		LN			Term	>7	3.1	no		2
27	31	V	151	57	24	67	yes	no	G2P1L1		no		Repeat CS		WD	Term	>7	3.8	no		7
28	27	IV	153	55	24	65	no	no	G2P1L1		no		LN			Term	>7	3.2	no		2
29	28	V	155	53	23	63	no	no	G2P1L1		no		Primary CS	CPD		Term	>7	3.3	no		2
30	35	V	157	51	23	61	yes	no	G2P1L1		no		Primary CS	CPD		Term	>7	3.4	no		7
31	27	IV	158	52	22	62	no	no	primi		no		LN			Term	>7	2.2	yes	Meconium	7
32	25	IV	151	50	24	60	no	no	primi		no		LN			Term	>7	1.7	yes	IDM	2
33	33	IV	145	48	21	58	no	no	primi		no		LN			Term	>7	2.8	no		2
34	26	IV	166	44	21	54	no	no	primi		no		LN			Term	>7	2.8	no		2

INSTITUTIONAL ETHICS COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
CHENNAI-10

Protocol ID. No.05/2017 Meeting held on 03.03.2017

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "**Maternal and Fetal Outcome in Obesity Complicating Pregnancy-A Prospective Cohort Study** " submitted by Dr.K.Padma Bharathi, M.S. O&G, PG Student, GKMC, Chennai-10

The Proposal is APPROVED.

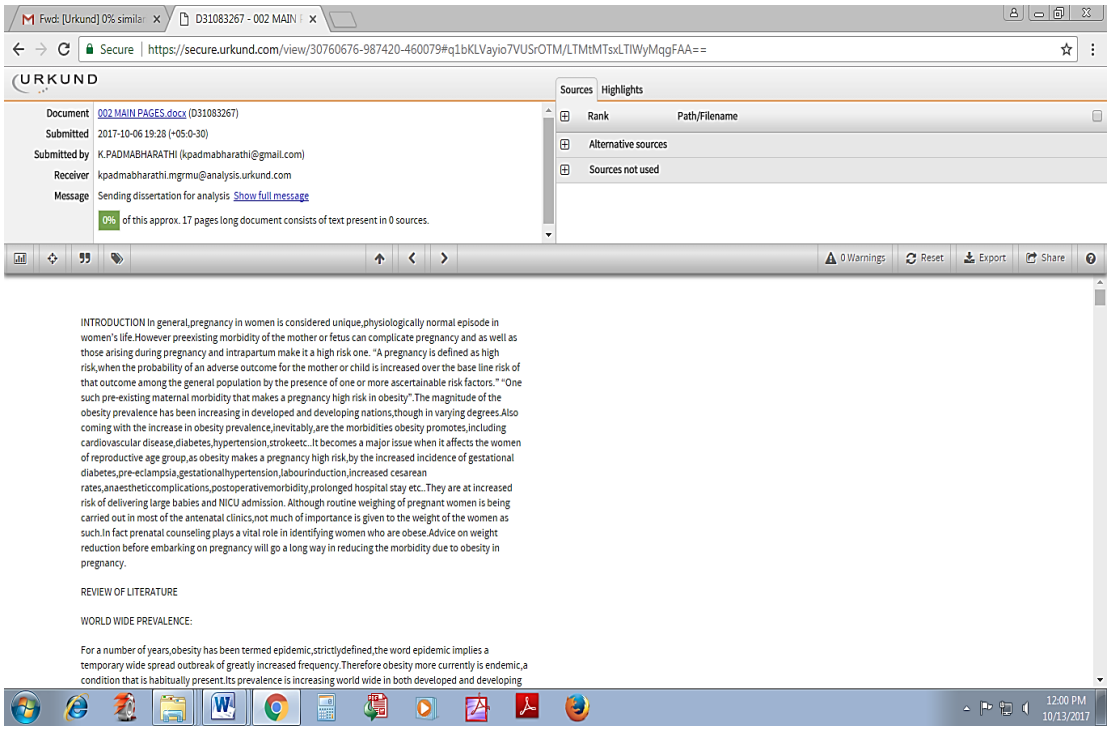
The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

22/3/17

DEAN
Govt. Kilpauk Medical College,
Chennai-10.

22/3/17

My Comp<E Colon< ME 1 Sec> Ethical Committee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INTRODUCTION In general,pregnancy in women is considered unique,physiologically normal episode in women's life.However preexisting morbidity of the mother or fetus can complicate pregnancy and as well as those arising during pregnancy and intrapartum make it a high risk one. "A pregnancy is defined as high risk,when the probability of an adverse outcome for the mother or child is increased over the base line risk of that outcome among the general population by the presence of one or more ascertainable risk factors." "One such pre-existing maternal morbidity that makes a pregnancy high risk in obesity".The magnitude of the obesity prevalence has been increasing in developed and developing nations,though in varying degrees.Also coming with the increase in obesity prevalence,inevitably,are the morbidities obesity promotes,including cardiovascular disease,diabetes,hypertension,strokeetc..It becomes a major issue when it affects the women of reproductive age group,as obesity makes a pregnancy high risk,by the increased incidence of gestational diabetes,pre-eclampsia,gestationalhypertension,labourinduction,increased cesarean rates,anaestheticcomplications,postoperativemorbidity,prolonged hospital stay etc..They are at increased risk of delivering large babies and NICU admission. Although routine weighing of pregnant women is being carried out in most of the antenatal clinics,not much of importance is given to the weight of the women as such.In fact prenatal counseling plays a vital role in identifying women who are obese.Advice on weight reduction before embarking on pregnancy will go a long way in reducing the morbidity due to obesity in pregnancy.
REVIEW OF LITERATURE
WORLD WIDE PREVALENCE:
For a number of years,obesity has been termed epidemic,strictlydefined,the word epidemic implies a temporary wide spread outbreak of greatly increased frequency.Therefore obesity more currently is endemic,a condition that is habitually present.Its prevalence is increasing world wide in both developed and developing
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